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TOXIC GOITRE.[1]

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I SHOULD like first to express my deep appreciation of the valiant efforts of those who have spoken before me tonight. I learned quite a lot from my friend, Dr. Wright Smith, and I may say that I have been forced to conform by clinical observation to the view that he holds that all the toxic goitres can be included under one heading and as different degrees of the one state. I am indebted to Dr. Sewell for his masterly theorization, which, by the same token, has an undercurrent of sound thought, because, whereas my mind on the subject before was a blank, it is now somewhat of a tangle. I think a tangle is better than a blank, because it stimulates one towards clarity of thought.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 7, 1931.

Historical.

Now Graves's disease, as diseases go, is by no means an "old timer", because, as far as I know, Parry described it in 1825. Graves, who was perhaps more academic, described it fully and recognized its importance, and almost concurrently Basedow described it in 1840. Therefore, the disease is young enough and I am old enough to be tempted to be somewhat reminiscent in my remarks. And I recall when, as an enthusiastic young student in Adelaide, I appealed to my teacher of those days, a clear-eyed, cultured gentleman, as to whether I could start work in the wards, and he said with a smile, which I came to understand later, "Oh yes, Mr. Smith, there is a case over there". It was a "heart" case, and as I walked towards the patient I was met by her eyes, which

literally popped out to greet me. I remember with what strange interest I investigated her condition. My enthusiasm at the end of about six months' attention on her in the ward began to wane. I had assailed her ills with all my proud and new knowledge gained in the therapeutic lectures. The meagre and tardy results proved, far better than would success, a most valuable lesson. That she died was a regret to me, and I have often pondered on her probably better fate had she lived in these days of surgical treatment.

My early days of practice were spent in Gippsland, and I carried with me memories of the assault on the disease by a surgeon at the Melbourne Hospital, that sturdy, courageous man, William Moore, who had attacked it like every other disease that was assailable by surgery, and who had achieved a measure of success. But as he was a general anæsthetic man and knew in those days nothing but chloroform, you can imagine that his statistics for operations on toxic goître would not bear comparison with those of Dunhill. All the same, we have to admit that, if he did not hit the target of cure, he staggered it; because from amongst those that he chose to operate upon, we students saw a few in which the result was most satisfactory; dramatic in fact. Indeed one lady on whom he operated is still alive in Melbourne and is at the head of a very successful business. Moore's courage did not bar him from performing an adequate operation. As I remember it, he removed not very swiftly, but very surely, both lobes, and as often as not left the isthmus only. You can understand that he had tetany following in his train, but by some clever dexterity he appeared to avoid nerve injury. Nevertheless I went to Gippsland with this thought back of my mind, that if I met toxic goître there, I certainly would not advise operation. I had learned from Trousseau to look out for the early cases, and am comforted to think that I turned in the right direction many of that type.

I can recall one graver case that we would now place as well established and over the six months' duration. With optimism, perhaps with iodine, and with very much rest, I had carried her to a stage of being a stone heavier, and, as I thought, well on the way to a cure. Her people, however, were dissatisfied and insisted on her visiting the city because of this persistent lump in her neck, which, by the way, became hard, and, as you all know, less active. I heard nothing for a few days, and then the village crier went forth, and I heard that Miss So-and-So was dead. When she left, she was in what I considered the pink of health. I did not need to ask any further questions, but in due course it came back to me that she had been operated upon, not by Moore, and that she had been given a general anæsthetic, which I judged to be chloroform.

Now at this particular period you must remember that Moore came very near to making a success of toxic goître treatment because he was urged to use the local anæsthetic, and, although we as house surgeons did not then know why we were so wise as to advise that, he was the type that was never fond of it. It was brought to the table for him, and the anæsthetist was there also, and when, as you have often seen, the patient displayed in acuter form the symptoms so disturbing to everyone, he would say: "Oh come on, please give her chloroform." Now Dunhill, knowing all this, and with that stout heart of his and, as I have always thought, a constant endeavour towards one honourable end, charged himself with the soul strain of local anæsthesia in such cases, and, as you all know, had dramatic success. Some of his earliest cases being in the stage of cardiac decompensation, the secret of his success was this, that he had local anæsthesia. He was soon thereby emboldened to approach every case. He approached bad ones and he performed single-sided operations, the natural thing to do as a final desperate attempt to save life. He did not cure all the patients, and he very rapidly awoke to the conclusion that he was not removing enough of the gland. He thought: "If I can attack this once, I can attack it again"; hence the second stage operation, still under a local anæsthetic. You must remember that in our journals here in 1908 there is a record of his in which there are thirty-five cases given and, summarizing these, he set forth the conclusion that it was necessary to remove more than a single lobe, and in this vital point he led the world. It is only fair to say that at this time Kocher had performed 200 operations on thyreotoxic goître with about 5% mortality and approximately 80% cures, but he was very careful to say in his articles that he removed only one lobe and the isthmus, and did not attack the very serious fibrillating cases that Dunhill attacked at the very beginning. He (Kocher) classed them as inoperable. The literature of the Americans also contains no statement about removing more than one lobe before 1915, when, in reviewing the whole situation, Judd says that "those cases not cured by a one-sided thyroidectomy would be rendered very much better if some of the remaining lobe were removed". Berry, the leading surgeon on goître in England, who had performed many thousands of operations, had at that time (1913) performed only 55 operations on thyreotoxic goître. Moreover, in that year in London, 97 patients with thyreotoxic goître were operated upon in all the general hospitals with a death toll of 16; of that number 55 received chloroform and a small number, about 15 or 16, a local anæsthetic, the others presumably being given ether. On the other hand, by far the greater number of Dunhill's goître operations had been on patients with toxic goître. It is interesting to note that about 4% only of all goîtres in Switzerland are of the toxic type, whereas in this country it looms as the paramount condition of the thyreoid gland demanding surgical treatment. Now from the local anæsthetic in the severe case Dunhill's evolution was such that he found that the strain on himself and on the patient with a local anæsthetic was so great that he was tempted to use a little ether in conjunction at the critical period of severest manipulation. From that there was an evolution to the use of ether alone in milder cases; and I can remember at that time myself using rectally administered ether anæsthesia. I can recall a series

of six or seven operations that I did at Saint Vincent's Hospital with delightful ease, but at that particular time one heard here and there of a disaster with ether given by the rectum, and so I just quietly refrained from using any more. For a few years there was a see-sawing between a local anæsthetic, a local anæsthetic *plus* ether, and in other cases pure ether, until the advent of gas and oxygen, which at the present day stands with me as first choice, except for the very grave cases, in which local anæsthesia is still preferred. So much, then, for the anæsthetic and the history of it.

History of the Operation.

One turns now to the operation itself. We are told that as far back as the tenth century a gentleman by the name of Albucasis performed in Bagdad an operation for the removal of a goître, and, as the historian says: "He well knew how to check the hæmorrhage with the ligature and the hot iron." Passing from those mediæval times, we have an account of a surgeon in England in 1770 who "accomplished extirpation of the thyroid gland with the result", as the historian says, "that for several days and for several nights different people pressed upon the neck in order that the hemorrhage might be controlled". And by 1800 it was agreed that many surgeons had accomplished this operation quite well. The most notable performance was that of Hedenus in Berlin, who at about that time gave an account of having removed six large suffocating goîtres without much hæmorrhage and with no deaths. We are inclined to say of these surgeons, "stout hearts", and I might add the same of the patients also. Then, as time went on, there were increasing numbers, but the death toll was great. There were sepsis and hæmorrhage, you see, and it is curious to note that there is no history in that time of much strumipriva and very little tetany. The reason was that the death toll was great, and, in addition to that, the method of removal was by mass ligatures, which left the posterior portions of the gland unapproached. Then came Simpson, Spencer Wells and Lister, and, following on their epoch-making contributions, the development of the modern operation; Kocher, with his hundreds of operations, and Billroth very much the same, and both encountering much *cachexia strumipriva* and tetany in their results. We then have Mikulicz publishing in 1886 a series of cases in which he described the leaving of the posterior portions of the gland in performing thyreoidectomy, thus avoiding the usual complications. It must be noted, however, that he still thought that cachexia and tetany were due to nerve interference and not due to the removal of vital organs. That was left for the British physicians to point out at a later date. Thus it is seen that we owe the modern type of operation to Mikulicz.

Medical Treatment and Operation.

Now I am rather surprised and not a little gratified that in Dr. Sewell's address there was no mention of protracted medical treatment of thyreotoxic goître. There is no mention of one or even two years of "skilful neglect". All the same, by one means or another, it happens that there is a waiting period, and this waiting period is very often not guided by any medical means, and it happens that in many instances a patient is not seen at all until the condition is well developed. I think the most aggressive surgeons admit that there should be a waiting period; that is to say, a period in which there is rest, in which, as Dr. Sewell ably points out, there is a reasoned attempt to counteract the extra tissue waste by good food in plenty, by iodine, and, in severe cases, by the insulin extract. It has been said that the ætiology of toxic goître may be summarized as "sex and sepsis". Like many catch phrases, that is only a part, not the whole, truth. If by "sex" is meant emotion, normal and abnormal, both in excess, then we have a useful indication for treatment during the medical period of management. This is the period in which optimism is so helpful. One writer believes that it is more valuable than iodine, though in fact I believe both are very necessary. The anxious typist with the widowed mother must be relieved of her load in very fact by others able to bear it; the working man's wife must have her children placed in other dependable hands; the exophthalmic taxi-driver with a family must have his richer brother's financial aid. The physician cannot strive for these too strongly, nor can he ever too loudly proclaim cure possible. It has also been said that this "sheltered life" treatment may go on in the well-to-do until medical cure arrives, even if it be two years or more. I am amongst those who strive for finality as early as may be, and I cannot see why the well-to-do should be treated in any other way than the poor. There is one best way, and that should apply to all. The lady who has all leisure and can lead what is called a sheltered life, does not desire to lead a sheltered life as a rule, and if there is any remedy which can be brought to bear, which is safe, and which offers a high probability of rapid cure, surely she should have that just the same as the typist who must work. It is one's custom, therefore, to advise operation in any case existing six months or more in which there are appreciable signs of the disease not rapidly improving. Incapacity for work, even when intermittent, is still an indication for surgical operation. Further, there can be no discussion about the need for operation in the third stage, when cardiac decompensation has set in. Dunhill has shown us the possibility of surgical operation in these and the good results accruing in otherwise fatal cases.

Very well, now, what has to be done in the way of an operation, because I take it that this is the surgical side? Dr. Sewell has said that in the treatment of sepsis he prefers to have the operation on the goître go first. I do not think that most workers are in accord with that, unless the condition is so grave as to dominate all other considerations, but they would not have his reason for disagreeing with it. They say that it is desirable

to have the patient in as good a condition as possible before the operation, and it has been my habit (and I think most agree) that, if easily removable sepsis is present, it should be treated and cured before the goître operation is performed. In this age of "atonsilosis" I suppose it is not necessary to mention that the septic tonsil should be removed. The problem that I have had to meet more than once is whether some organ, such as an overgrown succulent fibroid toxic uterus, should be removed before or after the goître, and I should like Dr. Sewell's opinion on such a condition as that. Then it has been said that the word "sex" was used. I presume that means all emotion, and, seeing that most of the patients are women, it cannot follow that it can be applicable entirely, because I do not think that emotion is more developed in the one sex than the other. A definite attempt, however, should be made to remove all emotional stress from the patient for a period before operation, just as has been insisted on in the medical treatment, and I think that is a very good point. It is not subscribed to by all, because I have been rung up more than once by medical men to know whether they could go to see the patient. Then there should be perfect team work surrounding the patient at the time of operation. I think it is very desirable that she should have the utmost confidence in the undertaking, the utmost confidence in all those that are around her. It has been said by many that she should not even know when the operation is to be. I think that is a thing to be thought of, but at the same time not always adhered to. Elaboration on that idea need not be made.

The Anæsthetic.

As to the anæsthetic, what is to be used? I have a feeling that in the approach to the very serious case a local anæsthetic should be essayed at any rate, and in this matter it is a case of *tot homines quot sententiæ*. Some prefer, as a preliminary soporific, "Amytal", while others will tell you they have had great success with hyoscine and morphine. I have for years used hyoscine and morphine, combined with a local anæsthetic, and it has been the custom to go right through the operation without having to call on the general anæsthetist who has been present; and frequently after operation the patient would ask whether it had begun. Very many of them on the next morning could not remember anything about it. Incidentally they have complained in an automatic way at the time of operation about pain and so on, but there is no memory of it. All the same, in a case that is not an entirely anxious one, I believe that the approach along the lines of one of the hypnotics *plus* gas and oxygen is a very satisfying and effective means of getting through the trouble. As to the age of the patient in guiding us in our actions, the question is raised: Should the very young be subjected to an operation in the form of a subtotal removal?

The youngest that I have met is a child of five who was ill and remained ill for a period of eighteen months because of this fear that early removal of a large part of the gland might lead to maldevelopment. So far as this patient is concerned, operation has been performed now two years, and she is very well. She has grown quite well and shows not the slightest sign of delayed development.

In the very old we have to remember that it depends entirely on the type of case. Occasionally one sees a primary thyreotoxic goître in an oldish person, and one would approach this surgically with extra caution. On the other hand, one has to remember that in the older patients the so-called secondary type is the rule, and therefore the toxic action is not so great. If the nervous symptoms are not well marked, I am inclined just to treat the patients as "ordinary risks".

The Operation.

A few words then about the operation itself. I do not suppose any surgeon wakes up and fails to remember when he has to do an operation on a toxic goître. Rather he wakes with verve and issues forth, as one might say, on his toes, because he knows that at every stage of the performance he has to be alert and well armed in every way. No operation needs such careful team work as does one on a toxic goître. The assistant should be expert, and should be able to do almost anything that the operator himself can do. Not only that, but he should, if possible, be used to the methods of the operator. The anæsthetist as a rule in these days is a man who has seen many of these operations, and in my opinion he should be a specialist anæsthetist.

The surgeon approaches the patient with the idea that in a severe case he may not operate that day, and the question is: If he does operate, what is he to do? There are stages of aggression on the neck in thyreotoxic goître. The very worst of them, I believe, even now should be approached by a ligature of the arteries as a first step. To show how the psychology of the surgeon will affect his whole attitude towards the operation, I have known Dunhill to approach a case with the idea of tying the thyreoid arteries as the sole attack for the day. That part of the operation has gone well, and then, with an immediate decision that he could hardly explain himself, he has said: "I will go on." He has extended the incision and had one side of the thyreoid removed in a very short time. Then he has hesitated and asked the anæsthetist something. Then he has said: "I will finish", and before much longer the suitable portion of the remaining lobe has been removed, and the patient subsequently did quite well. That experience really sums up the attitude of the surgeon towards the operation. Many of his actions become subconscious. They are all directed towards one end, and should be as swift as possible compatible with safety. I believe the man who never stops and never hurries finishes the goître operation first. There is always the fear of hæmorrhage, and there is always an automatic response to each type of hæmorrhage. One type will be met by simply leaving that side of the operation with a firm gauze plug in place. Another one will

be met by under-running with silk, because it is impossible to leave a clamp in that position. I detail these things because I wish you to appreciate the surgeon's attitude of mind towards the attack.

The incision in the neck should be made almost transversely, that is to say, like a collar, and parallel to the creases of the neck. It should be ample, because in that position it can be restored without leaving a bad scar. I do not know that surgeons differ very much in the details of the operation, but there are differences to be observed in watching the method of approach of different men. One man is ruthless from the beginning and is determined to get right through to the base of operations as quickly as possible. There is a cut right across the neck, ruthless division of the muscles, ligatures are put on all the arteries of both sides, and the gland is open to inspection by all. On the other hand we have a man like Rienhoff, who makes a small incision. He does what looks to be a very difficult operation, but what in his hands no doubt is quite easy. Personally I approach the operation with a perfectly open mind. I am sure there should be a generous skin incision. I am sure that the upper flap should be reflected well above the thyreoid cartilage and that the lower flap should be released thoroughly also, in order that approach below may be easy. It is not my habit as a routine to divide the smaller muscles, because it is possible in many cases to deliver the lobe without difficulty, and if that is not done, then so much time is saved. I think every surgeon then, having exposed the muscles of the neck, makes an incision vertically in the mid-line through the cervical fascia.

Let me digress for a moment to say that I have done a few by attacking each lobe through the intervals between the smaller muscles and the sterno-mastoid. That is a very easy way, but that approach sometimes involves leaving the isthmus.

As a rule, however, the mid-line incision in the fascia is made. It is very often a difficult matter to recognize the plane surrounding the gland, but the very deepest nearest to the gland is raised in its turn and a space is entered which practically contains the gland itself. The interval thus displayed is enlarged with the knife handle and finger, and will extend quite readily until the finger, running easily upwards, extends behind the upper pole, and an artery forceps held in the other hand is made to invaginate its way through from the mid-line towards the forefinger, and thus the upper pole is exposed, clamped, and tied. Here ends the first stage of the operation, and here begins the critical one.

A sort of infighting then begins, in which clamps are applied serially and the tissue cut between them. The assitant follows this stage of the operation by supplying the knife and clamps in steady rotation. The operation proceeds. Although it appears to be by sight, it is also very much by touch. The separation between the trachea and the gland is recognized and proceeds piece by piece. So the battle goes. Then as this lobe is separated and the tissues are pushed from the lateral border towards the great vessels of the neck, piece by piece of the remaining tissue and vessels is clamped and cut with a knife until the whole of the lobe becomes free. Then the urgent consideration arises: Where is the recurrent laryngeal nerve? How can it be avoided? In later times almost everyone has striven to leave a slice of the posterior border which shields the nerve from damage. That danger past, there comes the anxious question: May the surgeon proceed to the removal of portion of the remaining lobe? What are the danger signs? The anæsthetist, who should be really a consultant at this stage, will say "yea" or "nay" from his point of view. He decides many things. His experience perhaps tells him that the patient is getting at a low ebb and the pulse is poor and rapidly increasing; or that there is more fluid in the trachea than there should be, or that the fluid is frothy, indicating right heart distress. The surgeon, on the other hand, says: "The patient has lost a good deal of blood," or: "Well, the patient has not lost much blood." The appearance of the patient goes for something, as also does the time that the operation has lasted. A rapid decision is made as to whether more should be done. Remember that if you go on and things go amiss, you have a very narrow margin of safety. I feel that if there is doubt, you had better not go on. "We must teach ourselves that honourable stop, not to outsport discretion." You must be prepared to run away and live to fight another day. In no part of surgery is that truer than it is of the decision at that stage. The next step is the rapid restoration of the structures of the neck. I believe it is an important point to restore the small muscles of the neck in the mid-line as completely as possible, because if you do not, there is a great tendency for the trachea to become adherent to the skin and to cause an unsightly depression.

I am a great believer in efficient drainage in severe cases. I believe that William Moore lost some of his patients because he did not use drainage at all, and I believe he saved some of his later patients because he drained very well. I believe that in a severe case, that is, one in which there is a post-operative crisis of a grave type, the patient might be helped if the wound were widely opened again. I have said that the post-operative stage is really the greatest crisis for the patient with thyreotoxic goître. You know that there are these crises from time to time during the course of the disease. They must be met with intensive iodization for a day or so. The post-operative one is the gravest of them all. The pulse rate in some cases rises even to 200; there are the pain and distress in swallowing, cyanosis, sweating, restlessness, insomnia, and the whole gamut of an imminent disaster. It is really remarkable how imminent that disaster is, and even more remarkable how often it does not happen. I do not know how some of these patients get well, and yet they do.

I am talking about a grave post-operative crisis. How is it to be met? I said it was to be prevented

by a rapid operation. I also said it was to be prevented by an operation in which there is very little hæmorrhage, if possible; but afterwards I think that fluid intake should be used by every known means to dilute the "toxins". It is my habit to have every patient receive a submammary saline injection, even on the table. I suggest that in these days, with our knowledge of iodine, in grave cases it might be wise to add some iodine to that submammary injection. I suggest that as a possibility, but I would definitely add iodine to the rectal saline solution and glucose injections that are being administered, because even after local anæsthesia there is probably some vomiting, and probably there is not absorption to the fullest extent of fluid given by the mouth.

The problem will always arise of the value of sedatives, but I must admit that when pain and loss of sleep are present, I feel that more harm is being done by not giving morphine than by giving it. I have seen nothing but good come from the generous use of oxygen in the crisis. I see some eminent cardiologists present, so I shall say nothing about the use of digitalis, except that before the patient dies some is usually given. In the matter of iodine, may I suggest that it be bruited abroad that if a patient is not doing well medically, we as surgeons pray you not to take the bloom off the case by using up the iodine response of the patient before she or he reaches the surgeon. I think that is a very fair request.

The Post-Operative Period.

The period of post-operative stress lasts from a few days up to ten days, but it is very soon obvious which patients are to do well, by the marked slowing of the pulse. The question is what to do later. It depends upon the degree of immediate response. Some of the patients are almost immediately cured and resume normal life quite soon, but I think on the whole it is better to regard an operation as just an incident in the treatment of thyreotoxic goître. It puts the patient on a better wicket, so to speak. Very few of them have been so recently ill of it that they have no harmful effects to overcome. Dr. Sewell has hinted that perhaps the patient with severe goître will always have a limited reserve. I see not infrequently patients who had operations many years ago. I remember a patient with a malignant breast only a few months ago. It was curious in this case—one of Dunhill's—where there had been apparently complete recovery, how, under the stress of a surgical operation, the patient repeated the rapid pulse and the restlessness of her former condition. This rather suggests that the underlying nervous state of which Dr. Sewell has spoken, persisted after she was apparently cured. There are other patients who received surgical treatment and who have only a narrow margin of reserve.

I recall a taxicab driver who was treated by me in Saint Vincent's Hospital in a two-stage operation and apparently cured. He reappeared years afterwards, again thyreotoxic, that is to say, thin, with damp skin, tired, and unable to work. It was suggested that he required a further operation, but it is interesting to note that he was sent home and told to go to bed and take Lugol's solution. He returned in about two months, much heavier, with good colour, no longer sweating and tremulous, and apparently quite well, although I doubt very much whether he has much capacity for really hard work.

Summary.

To sum up, we have a disease known to man, or described by man, for only a hundred years. In that time it has gone through various stages so far as our knowledge of it is concerned. My first text book told me that it was "a functional disorder of the circulation, probably arising from irritation of the cervico-sympathetic". Then it reached the stage of being regarded as partly surgical, and is now looked on as entirely surgical by many, and, when advanced, by all, with the results that Dunhill can now show a series of over 2,000 cases with a total mortality of 2·7% and a death rate of well under 10% in the severe cases which were regarded as inoperable formerly, and when the patient was likely to die very soon under any other treatment. We have reached this stage, but at what a cost to the patient and at what a cost of time and strain in surgery! I feel, from a more or less historical view of the matter, that if such great advance has been made in so comparatively short a time, a still greater will be made in the next fifty years, and I cannot help thinking that some other way out of the trouble will come than by what, after all, and good though it be, is only a surgical empiricism.

DIATHERMY IN THE TREATMENT OF GENERAL PARALYSIS OF THE INSANE.[1]

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IT is established that patients suffering from general paralysis of the insane improve greatly, even recover, when treated by fever produced by malaria. Before the use of malaria was discovered, it was known that occasionally general paralysis was cured or became quiescent for years after an attack of an acute illness, and patients treated by the Swift-Ellis method, which was generally accompanied by fever, also did well.

My attention was called to the fact that general paralysis was being treated in the United States of America through the medium of an artificial fever produced by the diathermy current. Mr. Trainor, of Watson and Sons, Limited, was able to let me have some literature upon the subject, and very kindly lent a diathermy machine, which has been employed in the treatment carried out at the Parramatta Mental Hospital.

The literature supplied consisted of papers by J. Cash King and Edwin W. Cocke, who read a

[1] Read at a meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association on April 21, 1932.

paper upon the subject before the Southern Medical Association at Miami, Florida, in November, 1929; also a paper by C. A. Neymann and S. C. Osborne, of Chicago, on "The Treatment of Dementia Paralytica, with Hyperpyrexia produced by Diathermy" (*The Journal of the American Medical Association*, January 3, 1931).

As the treatment was based upon advice and instruction contained in these two papers, the authors must forgive me for freely quoting from them.

King and Cocke administered eight to twelve treatments to each of twelve patients, but in another eight cases abandoned the treatment for reasons not given. They state as follows:

> After a lapse of from two to ten months after treatment we found eight of the cases showed definite improvement. Two of these had a clinical remission. Eleven of the twelve have shown a gain in weight and an improvement in their general physical well-being. One patient died of a complicating acute nephritis. This developed at the end of the eighth treatment.

Neymann and Osborne treated twenty-five patients in the Cook County Psychopathic Hospital. They sum up their results as follows:

> We have been critical in judging the results obtained, the more so because the results are so unusual. We divided the patients into three groups: (1) First were those that made absolute social adjustment and were able to maintain themselves outside the institution without supervision. This embraced sixteen cases, or 65% of the entire number. (2) The second group were decidedly improved and were able to maintain themselves with supervision at home. This group consists of two persons, or 8%. (3) The unimproved group, seven in number, five of whom were totally deteriorated when treatment was begun. From past experience with malaria and tryparsamide, we believe that dilapidated and deteriorated patients never recover when this deterioration has extended over a period of years.

The idea of the treatment is that by the production of a hyperpyrexia results will be obtained in cases of general paralysis of the insane similar to those produced by malaria; certain advantages are claimed by those who advocate the use of diathermy. It seems that by diathermy, if large electrodes and the necessary ampèrage are used, the temperature of the body can be raised to any desired degree, and with care and management, this temperature can be maintained at the will of the operator. The objective is to produce an abrupt elevation of temperature, as in malaria, to about 3·3° C. (6° F.) above normal. The temperature was then permitted to return to normal. These elevations were brought on each day for eight to twelve days.

In Neymann and Osborne's cases the number of treatments per patient varied from six to forty-nine, an average of fifteen. Treatments were administered twice a week. At first they sought to produce temperature curves analogous to those produced by malaria, but later attempted to keep the temperature above 39·7° C. (103·5° F.) for at least five hours, though some patients maintained this temperature for nine hours. In one of their cases the final temperature reached 42·5° C. (108·5° F.) without ill effects.

King and Cocke describe the technique of producing fever by diathermy as follows:

> A method of getting sufficient electrode surface to pass the current through the body without producing burns was a problem of greatest importance and was solved with no little difficulty. For this we now use a specially constructed jacket, which is fastened round the chest and abdomen by elastic straps. This jacket holds a seven by nine inch tinfoil electrode on the front and back of the chest and abdomen, thus making our electrodes practically cover the anterior and posterior surface of the torso. If these electrodes be applied with reasonable care, no difficulty will be experienced in passing more than sufficient current through the patient's body to obtain any rise in temperature compatible with life without producing even the slightest blister on the skin.
>
> To prevent excessive heat dissipation, the patient is carefully wrapped in rubber sheets and blankets. Rubber sheets have been found necessary because of the great amount of perspiration. The blankets and rubber sheets are arranged in the following way; a rubber sheet is spread over the bed on which the patient is to be treated, then a double blanket is spread over this sheet, which prevents puddling of the perspiration on the rubber sheet. Another blanket covered by a rubber sheet and two more heavy woollen blankets are thrown over the patient. Too much stress cannot be placed on the necessity of using plenty of blankets, for these serve as insulation against the escape of heat from the patient's body.
>
> With the electrodes applied to the body of the patient and the insulating material described above in place, the high frequency current is passed through the deep tissues. The current is gradually increased, beginning at zero, and may be allowed to reach its maximum in twenty to thirty minutes. The maximum current depends primarily upon the size and physical condition of the patient. It also depends on the shape of the temperature curve which the operator desires to produce. The amount of current required for practically any temperature will fall between 3,000 and 6,000 milliampères.

The Treatment and its Effects.

The diathermy machine lent by Mr. Trainor is of the open tungsten spark gap type, furnished with milliampère meters and adjustments for frequency and voltage. Four gaps would appear to be necessary in order that the heat generated within the machine should be dissipated. This is due to the fact that diathermy apparatus must produce high milliampèrage in this work, and for unusually long periods, continuously.

Twelve patients suffering from general paralysis of the insane have been treated with diathermy; the cases have been of various type and duration. Three of the patients were women and nine men.

Endeavours were made to maintain a temperature of over 39·4° C. (103° F.) for five or six hours per treatment and to give sufficient treatment to insure that the patients would have a total period of forty-two hours with a temperature above 39·4° C. (103° F.), as advised in treatment by malaria. In one or two cases this period was exceeded, one patient having a temperature of above 39·4° C. (103° F.) for seventy-seven hours, and two others for over sixty hours.

The control of the temperature and the control of the patient have had to be learnt by experience. In one or two early cases the temperature rose to somewhere near 41·6° C. (107° F.) and, although disturbing symptoms occurred, no lasting ill effects resulted.

The routine has been as follows.

On the morning of the treatment the patient is given an enema and put to bed. The electrodes are very carefully adjusted, bound in place by felt. The patient is then insulated by rubber sheets and blankets, as advised by King and Cocke. Nine blankets and three rubber sheets are used, one rubber sheet next to the mattress and two blankets underneath the patient. An air cushion is placed under the sacrum, ring pads under the heels, and a cradle over the feet. The patient is then wrapped in two blankets, outside which are two rubber sheets and seven to nine blankets placed over the patient.

At first electrodes of tinfoil supplied by Mr. Trainor were used. Later, on Mr. Trainor's advice, lead electrodes were employed; these were easier to apply and lasted longer. As, when the temperature rises to about 38·9° C. (102° F.), the patient becomes extremely restless, the additional precaution was adopted of placing the patient in a canvas jacket or camisole, which restricts the movement of his arms. If restless, the patient is apt to displace an electrode and may become burnt. The first patient obtained a slight burn upon the neck, by moving with her hand the back electrode. No burn occurred after the use of the camisole was commenced. No patient objected to the camisole, which has the further advantage of adding to the insulation. In winter it was found necessary to warm the room, as in a cold room it took from four to six hours to raise the patient's temperature to 39·4° C. (103° F.), depending partly upon the size of the patient; the lighter the patient the quicker the rise. In the summer months the same result was achieved in half the time.

After the electrodes are in place, the current is turned on, commencing with a low milliampèrage, which is raised to 3,000 milliampères in half an hour and left there until the patient perspires freely. Any higher milliampèrage is likely to burn a dry skin. The temperature desired is generally reached with a milliampèrage between 3,500 and 3,800.

When the temperature approaches 38·8° C. (102° F.) the patient has extreme discomfort and becomes very restless. He then receives a hypodermic injection of 0·016 gramme (a quarter of a grain) of morphine and 0·00065 gramme (a hundredth of a grain) of hyoscine. Some of the more nervous patients are given this at the commencement of the treatment and during the treatment, if necessary. About now the patient sweats freely, he is encouraged to drink warm water, and the milliampèrage is increased to between 3,500 and 4,000. The temperature is allowed to rise until it reaches between 39·4° C. (103° F.) and 40° C. (104° F.). The current is then turned off, but the temperature continues to rise for about another degree Fahrenheit. At this stage patients behave differently. As a rule the temperature rises to 40·5° C. (105° F.) or a few points above, and remains there for as many hours as desired, that is, until the patient is uncovered or partly uncovered, when it commences to fall. The rate of fall can be partly controlled by the rate of removing the blankets or by sponging. In some of the cases in the series reported here, a temperature above 39·4° C. (103° F.) has been maintained for seven hours; but as a rule the period has been limited to between five or six hours, as the more prolonged temperature seems to exhaust the patient. The intervals between the treatments have varied, but intervals of two days have been aimed at; these have been increased to three, four or seven days. The total number of treatments is usually seven or eight, but as many as thirteen have been given.

If the temperature falls before it is desired to fall, it can easily be raised again by turning on the current for from thirty minutes to an hour.

After having the morphine the patient is, as a rule, calm and quiet until the end of the treatment, but when waking, complains of much thirst, which continues for some hours. Although the temperature may occasionally begin to fall too soon, on four occasions it has risen alarmingly some hours after the current has been cut off.

In the first case the current was not turned off until the patient's temperature was 40·5° C. (105° F.); two hours after it reached 41·5° C. (106·8° F.). In a later treatment given to the same patient, the current was turned off when the temperature was 39·8° C. (103·6° F.); two hours later it had risen to 41·1° C. (106° F.). In another case, during the patient's seventh treatment, the current was turned off when the temperature was 39·9° C. (103·8° F.); the temperature kept even for four hours and then rose to 41·3° C. (106·4° F.). In the third case the patient behaved well until the eighth treatment. The current was turned off at 11 a.m., when the temperature was 39·5° C. (103·2° F.). At 1 p.m. it was 40·9° C. (105·6° F.) and the patient was partly uncovered. At 4 p.m. it was 41·3° C. (106·4° F.). The patient was then uncovered and sponged; the temperature took three hours to fall to 38·8° C. (102° F.).

In each case in which the temperature has reached 41·1° C. (106° F.) the patient has become unconscious, the pulse has been thready, but not rapid in proportion to the temperature. The patient in Case XII was seized with intermittent generalized convulsions when his temperature was over 41·1° C. (106° F.). All patients responded to cardiac stimulants, sponging and uncovering, and none suffered more than a very temporary inconvenience.

It was possible to continue treatment in all cases, but patient X became very cyanotic during his first two treatments. Cyanosis did not recur at subsequent treatments.

While the treatment is being administered the temperature is taken in the rectum every fifteen minutes. Observations have also been made of the pulse, respiration and blood pressure, this latter only in a few early cases.

The pulse on the whole is more constant and less variable than the temperature. It rises and falls with the temperature, but not in proportion. When the temperature is 40·5° C. (105° F.) the pulse rate is between 120 and 130, occasionally 140 per minute, but when the temperature has risen above 41·1° C. (106° F.), the pulse rate has not risen

with the temperature. The respiration rate increases slightly with the temperature, but has not exceeded 26 per minute.

Neymann and Osborne made observations upon the blood pressure, blood count, and upon the blood calcium and chlorides, the carbon dioxide capacity of the plasma and the non-protein nitrogen and uric acid of the blood. They observed a rise in the systolic blood pressure and a fall in the diastolic pressure, and, after a series of treatments, a fall in both the systolic and diastolic pressures, which they found remained at a permanently lower level. In the blood they found an increase in the red and white cells and in the hæmoglobin, an increase in the quantity of uric acid and non-protein nitrogen, a slight increase in the calcium and chlorides of the blood, and a decrease of the carbon dioxide capacity of the blood. The serological changes in the cerebrospinal fluid did not correspond with the clinical improvement. They report a decrease in the number of cells in all cases but two, and a decrease in the intensity of the colloidal gold reactions. In these matters they have made more thorough examinations than we have been able to make.

I was not successful in making blood pressure observations, as it rendered necessary the partial uncovering of the patient, which interfered with the rise of temperature.

A slight decrease in the numbers of red and white blood cells was found after a series of treatments. In one case there was an increase in the number of red cells. In two cases (X and XI) a very pronounced decrease in the numbers of red cells and an increase of the size of the cells were observed.

CASE X: Five weeks after the last diathermic treatment, and when his discharge was being considered, the patient's left knee joint became swollen and later completely disorganized. Three weeks afterwards the hip joint on the same side became affected, the leg being rendered absolutely useless. He was suffering from Charcot's disease of the hip and knee joint; X ray examination revealed extensive bony change of the left side of the pelvis and absorption of the femoral head.

He then became intensely anæmic. At the termination of treatment his red blood cells numbered 4,550,000 per cubic millimetre, and leucocytes 7,500 per cubic millimetre; the hæmoglobin percentage was 90, and the colour index 1·0; there were 60% of polymorphonuclear leucocytes, 25% of small lymphocytes, and 10% of large lymphocytes. Five weeks later the erythrocytes numbered 2,350,000, and the leucocytes 6,000 per cubic millimetre; the hæmoglobin value was 65% and the colour index was 0·7. The size of the red blood cells, as measured by Pijpers's halometer, was, for the average large cells, 9·08 micromillimetres, and for the average small cells, 8·5 micromillimetres.

A month later, after treatment, examination of the blood revealed the following:

Numbers of Cells:	
Red blood cells, per cubic millimetre	3,459,000
White cells, per cubic millimetre ..	8,600
Hæmoglobin Content:	
Percentage	70
Colour index	0·9
Differential Count:	
Polymorphonuclear leucocytes ..	60%
Large lymphocytes	10%
Small lymphocytes	30%
Size of Red Corpuscles:	
Average large, micromillimetres ..	8·3
Average small, micromillimetres ..	7·4

CASE XI: A week after the termination of his treatment the patient suffered from an acute pleurisy with effusion. He also suffers from anæmia; his red blood cells fell in numbers from 5,300,000 to 3,450,000 per cubic millimetre, but soon became normal with ordinary treatment.

In every case a pronounced shortening of the coagulation time was observed while treatment by diathermy was being carried out. The shortest coagulation times were observed after the patient had been under treatment for two or three hours and before the temperature had risen to the degree required. After the machine was turned off, although the temperature was higher, the coagulation time was longer.

Samples of urine taken twelve hours before and twelve hours after treatment were examined for specific gravity, acidity, ammonia content and urea content. In every instance the specific gravity was raised and the acidity was greatly increased, as also was the ammonia.

A typical example is Case XII, in which the findings were as set out in Table I.

The blood Wassermann test was carried out in nine cases, before and after treatment. The reaction was altered after treatment in four cases, that is, it was changed from positive to partial positive or indefinite "negative", and in one case it disappeared.

The Kline test was applied in four cases, both before and after treatment, and in all four the reaction lessened in intensity. In one case it changed from + to "negative", in two cases from ++++ to ++, and in one case from ++++ to +++.

The findings in the examination of the cerebrospinal fluid were different from those of Neymann and Osborne.

In every case there was a decrease in the number of cells and a decrease in the intensity of the colloidal gold or bicolour guaiacol test.

The Wassermann reaction of the cerebrospinal fluid from nine patients was examined before and after treatment, and changes were reported in four; once from partial positive to no reaction, twice from positive to slight positive, and once from positive to indefinite "negative". In

TABLE I.

The Effect of Diathermy on the Urine.

Specimen of Urine.	Amount.	Specific Gravity.	Acidity.	Ammonia Nitrogen Content.	Urea Content.
Obtained twelve hours before diathermy ..	800 c.cs.	1·004	4 c.cs. decinormal acid per 100 c.cs.	0·00952 gramme per 100 c.cs.	0·5 gramme per 100 c.cs.
Obtained twelve hours after diathermy ..	300 c.cs.	1·020	48 c.cs. decinormal acid per 100 c.cs.	0·0076 gramme per 100 c.cs.	2·0 grammes per 100 c.cs.

TABLE II.

Condition of Patients Before and After Treatment, Accessory Treatment, et cetera.

Case Number.	Sex.	Age.	Weight	Type.	Blood Wassermann Reaction.	Kline Reaction.	Cerebro-Spinal Fluid.					Number of Diathermy Treatments.	Hours above 39·4° C. (103° F.)	Other Treatments.	Remarks.
							Wassermann.	Cell Count per cubic Millimetre.	Globulin Reaction.	Takata Ara Reaction.	Bicolour Gualacol Reaction.				
I	F.	40	st. lb. Before 8 13 After 8 3	Demented.	− −		+ −	2	+ −	+ −	1311100000 −	7	44¼	Intravenous injections of "914".	Made much mental improvement. From being dull, apathetic and lazy, became bright and a useful worker, and developed wit and a sense of humour. Has been out on leave of absence for a short period. Improved.
II	F.	32	Before: 8 5½ After: 8 6	Depressed.	+ +		+ +	70 15	+ +	+ +	Gold Sol. 5555854100 Bicolour Gualacol. 1243431100	7	42	Intravenous "914".	Extremely ill on admission, after operation for *fibromyomata uteri* and appendicitis. Became well and rational and was discharged four months after admission. A recent report states that she is remarkably well. Much improved.
III	F.	33	Before: 10 4 After: 10 8	Exalted.	+	++++	+ +	18	+	+	2443310000 0111¼00000	7	40½	Injections of "914".	Lost all her delusions of grandeur, became quite rational and apparently well. Discharged two weeks after termination of treatment. Much improved.
IV	M.	45	Before: 9 0	Demented.	+		+ Indefinite −	2	+ Partial +	+ Partial +	4444211000 −	10	61½	Injections of "Muthanol" (1·0); "Kharsulphan" (10).	Demented. No improvement, but became quiescent. Not improved.
V	M.	44	Before: 9 0 After: 7 9	Depressed.	+	+++	+	43	+	+	134444200	7	45	Injections of "Muthanol" (4), "Kharsulphan" (4).	After treatment was brighter and more communicative. Developed lobar pneumonia and died, three weeks after treatment. Not improved.
VI	M.		Before: 9 0 After: 8 7	Exalted.	+ +	−	+ Partial +	61 10	+ +	+ +	1444444420 1444443100	13	77	Injections of "Muthanol" (1·0), "Kharsulphan" (10)	Lost his delusions of grandeur and became indignant if riches and titles were referred to. Lost much ground and became apparently totally demented, but later regained some of his former health and passed into the fat and quiet stage. Not improved.
VII	M.	50	Before: 11 0 After: 9 7	Demented.	+ +	++++ +++	+ ·	43 0	+ +	+ +	24211100000 +	10	61¾	Injections of "Muthanol" (1·0), "Kharsulphan" (10).	Before treatment was demented, incapable of conversation; his mind was a blank and memory nil. Improved, became able to recognize those around him, and able to do his work of book-binding. Improved.
VIII	M.		Before: 12 6 After: 11 9	Demented.	+ Incomplete −	++++ ++	+ Partial +	15 11	+ Slight +	+ Partial +	344432100 0001210000	11	64	Injections of "Muthanol" (7), "Kharsulphan" (7).	A very acute case. Symptoms had existed three months before admission. Demented, depressed and completely disorientated. Improvement slight, apparently arrested. Not improved.

TABLE II—*Continued.*

Condition of Patients Before and After Treatment, Accessory Treatment, et cetera.

Case Number.	Sex.	Age.	Weight	Type.	Cerebro-Spinal Fluid.							Number of Diathermy Treatments.	Hours above 39·4° C. (103° F.)	Other Treatments.	Remarks.
					Blood Wassermann Reaction.	Kline Reaction.	Wassermann.	Cell Count per cubic Millimetre.	Globulin Reaction.	Takata Ara Reaction.	Bicolour Guaiacol Reaction.				
IX	M.	45	st. lb. Before: 11 9 After: 11 7	Exalted.	+		++	130 0	++	++	4444443330 1444443100	8	45¼	Injections of "Munethanol" (10), "Kharsulphan" (10).	Improved very much, lost his delusions and had good insight into his condition and was discharged a month after treatment. Much improved
X	M.		Before: 9 8 After: 9 7	Depressed.	Incomplete	+	Slight +	60 0	+ Slight +	+ Slight +	3444443321 0010110000	9	47¼	Injections of "Munethanol" (7), "Kharsulphan" (7).	Made much mental improvement, lost his depression, had good insight, and displayed an interest in life. Five weeks after treatment, became extremely anæmic. Developed an extensive Charcot's disease of hip, which later became ankylosed. Mentally, much improved.
XI	M.	48	Before: 8 11 After: 8 8	Exalted.	+	+ + + + + + + +	Indefinite —	70 4	+ Slight +	+ Slight +	344443321 0011000000	8	44¾	Injections of "Munethanol" (2), "Kharsulphan" (2).	After treatment had an acute pleurisy with effusion; ill for some weeks, also became anæmic. Lost his delusions and became more rational, but was for some time irresponsible. Later made decided improvement, became an industrious bookbinder (work new to him), and was discharged. Much improved.
XII	M.	34	Before: 8 4 After: 8 4	Depressed.	Indefinite	+	++	7 4	+ Slight +	++	4444431000 1344421000	6	33¼	Injections of "Munethanol" (2), "Kharsulphan" (2).	Improvement great, more cheerful, had an interest in games and work. Later, although cheerful and able to work, failed somewhat, having confusion of thought and hesitancy in speech, but was well enough to be discharged to return to America. Improved.

four cases the globulin test changed from positive to slight positive. The Takata Ara reaction diminished in three cases.

Although the patients while under treatment perspire to an extreme degree, the loss of weight was not great, not more than from 2·25 to 3·15 kilograms (five to seven pounds) in most cases, and was made up within a few weeks after the cessation of treatment. There was one exception, patient IV, who lost 8·5 kilograms (nineteen pounds) in weight.

Except in Case I, in which the patient received a slight burn, I have had no trouble with burns; but the possibility of such has to be constantly borne in mind, more especially as some of the patients are demented and are at some time during the treatment under the influence of morphine.

Pressure sores have caused trouble; neither King nor Neymann seems to have encountered these. Patient I had a sore over the sacrum and on both heels. Another patient also had sores on both heels and on one toe. This was attributed to the excessive sweating and remaining in one position for several hours. An air cushion is now used for the sacrum, ring pads for the heels, and a cradle to keep the pressure of the blankets off the toes. Since these precautions have been taken there have been no further pressure sores.

Summary.

It is difficult to summarize the results, as the term "recovered" cannot be used until a greater length of time has elapsed.

For the sake of illustration I will divide the results into three groups and describe them as follows: "Much improved" where the improvement has been great

and definite and where the patient has left the hospital or is ready to do so; "improved" where the patient has become brighter, more intelligent and able to work; "not improved" where there has been none or but slight improvement.

Five cases can be placed in the "much improved" group, namely, Cases II, III, IX, X and XI. Among these are two of the three women of the series. Case X is the case with the Charcot's joint. As this patient's mentality is decidedly better, he is placed among the "much improved".

Three cases, I, VII and XII, are included in the "improved" group.

TABLE III.

Summary of Results of Diathermy Treatment.

Mental Condition.	Case Number.	Result.
Demented	IV	Not improved.
	I	Improved.
	VII	"
	VIII	Not improved.
Exalted	VI	" "
	IX	Much improved.
	III	" "
	XI	" "
Depressed	II	" "
	V	Not improved.
	X	Much improved.
	XII	Improved.

Number of patients not improved = 4.
Number of patients improved = 3.
Number of patients much improved = 5.

The "not improved" cases number four, and include Cases IV, V, VI and VIII. The downward course of two demented patients in this group was apparently arrested.

Among the "much improved" patients were three exalted and two depressed patients; four of them had had symptoms of general paralysis for less than twelve months.

Two of the demented patients were much better after the treatment. In Patient VIII there was a slight change, in that he could read the paper. His downward course was seemingly arrested. Patient VI, who very quickly became demented after treatment, was an acutely maniacal general paralytic, a type that generally does well with malaria. Patient XII suffered from chronic bronchitis, which cleared up in a remarkable way after two treatments.

The length of time and the number of cases recorded are not sufficient to say that diathermy has any advantages over malaria, but in diathermy we have apparently a means of giving pyrexia to general paralytics, with results comparable to malaria. It has the advantages over malaria in that, by employing it, we are not dealing with or giving a disease, and no precautions against the spread of disease are necessary. It is more under control, more easily obtained than malaria, and always ready when needed.

The strains of malaria deteriorate with time and are apt to die out, as there are not always patients to carry the disease.

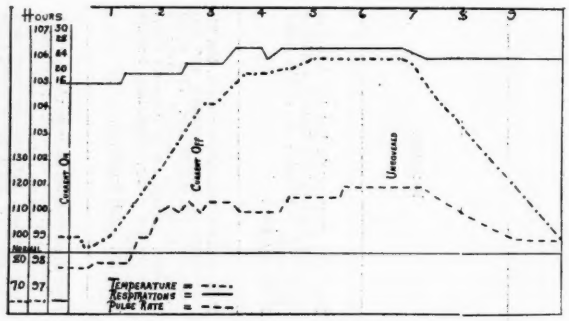

FIGURE I.
Showing a regular temperature, but a degree too high. Current not turned off until temperature was 104° F.

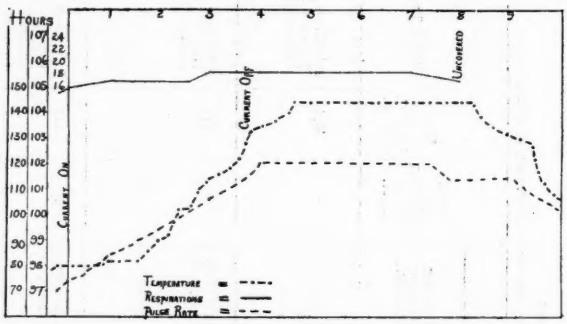

FIGURE II.
Showing a well maintained temperature after a slow rise.

Acknowledgements.

My thanks are due to Mr. Trainor, of Watson and Sons, whose kindness enabled the work to be done; to Sister Kathleen Newman, sister in charge of the X ray department, who has given the diathermy treatments, who has done the practical work connected with it, and who has made the blood and urinary examinations; to Dr. Oliver Latham and his staff, for the serological examinations and reports.

Appendix.

Since this paper was written, four additional patients, three male and one female, have received a complete course of treatment by diathermy.

There has been decided improvement in the mentality of four patients, and it is anticipated that they will all be discharged.

In these later treatments the aim has been not to allow the temperature to exceed 40·5° C. (105° F.) or to remain above 39·4° C. (103° F.) for more than five hours. The results with this method, as far as can be judged, compare favourably with the earlier ones, and the operator's anxiety is greatly lessened.

A PROGRESS REPORT ON THE ROUTINE PRACTICE OF MALARIA THERAPY.[1]

By CLIFFORD HENRY, M.B., Ch.M., Dip.Psych. (Sydney).

Introduction.

MALARIA therapy, although still empirical, is emerging from the purely experimental stage. We

[1] Read at a meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association on April 21, 1932.

can definitely state that in the majority of cases of neuro-syphilis so treated, the expectation of life, the capacity for work, the comfort of the patient, remission of mental symptoms and comparative restoration of physical well-being, are very considerably increased. Malaria therapy is therefore worthy of study, in that it is a distinct advance in psychiatry; and it is hoped that it may point the way to still more important discoveries.

The subject has been intensively studied at Callan Park Mental Hospital since November, 1926, and the routine practice, which the experience of these five and a half years has proved desirable, has been collected in this paper, which includes information given in the "Report on General Paralysis and Its Treatment by Induced Malaria", issued by the Board of Control for England and Wales. This report summarizes also the methods and results used and obtained in Austria, France, Germany, Belgium, Holland and Czecho-Slovakia, which are very similar to our own. Dr. O. Latham has kindly supplied some additional information.

Choice of Patients.

Any patient with any form of neuro-syphilis may benefit, however debilitated, provided there is no other gross disease, for example, serious renal or pulmonary disease.

Some authorities, including A. Paige (*New York State Journal of Medicine*, December 1, 1931, page 1441), provide us with a formidable list of contra-indications, including old age, pregnancy, chronic alcoholism, and rapidly progressive neuro-syphilis. These are unimportant. One of our patients, aged sixty-three years, improved remarkably; pregnant women improve twice as frequently as other patients, and rapidly progressing states improve as greatly as others, and there is all the more need for immediate treatment. A patient of mine with long-standing aortic regurgitation, treated five years ago, is in apparently robust health at Callan Park today.

The patients selected should not recently have been treated with preparations of arsenic, mercury, antimony, methylene blue or carbolic acid.

Procedure Before Infection.

Some workers advise a quinine tolerance test; but in a series of 395 cases at Callan Park it has not been found necessary, and it may be undesirable, as a trace of quinine in the system may interfere with successful infection. No undesirable effects from quinine have been observed at Callan Park. Should quinine intolerance be noted, malaria therapy should be tried, and "Neosalvarsan" should be used as a means of terminating the fever instead of quinine.

Blood grouping of patients should be carried out if intravenous injection is adopted, but is unnecessary if the inoculation is made by subcutaneous or intradermal injection.

Examination of centrifugalized urine for blood pigment by Heller's test should always be carried out before inoculation.

In addition to these, a blood film should be stained with Leishmann's stain, labelled and preserved; a blood count should be made, and the percentage of hæmoglobin estimated.

The result of urinary examination should be recorded and in any doubtful case the blood urea estimation also.

Blood sugar estimation should be carried out.

A note should be made as to the state of the several bodily systems: pulmonary, cardiac, gastro-intestinal *et cetera*. The reflexes and serological results should be noted, with a *précis* of the mental state. The high comparative death rate within a month or two of inoculation makes desirable: (*a*) a complete examination before treatment and notation of same; (*b*) unremitting attention during treatment; (*c*) great care during convalescence. It should be noted that a similar high death rate occurs soon after admission in untreated cases, because many general paralytics are admitted when almost *in extremis*.

An estimation of the basal metabolic rate, by direct observation or by Read's method, is useful for later comparison.

Infection.

Two cubic centimetres of blood directly drawn from the vein of a patient, known to be infected with *Plasmodium vivax* only, should be injected under the skin or, better still, intradermally. This method has proved far superior to any other.

Defibrinated malarial blood packed in ice has been sent by aeroplane from Sydney to Brisbane and has conveyed the infection even when, as noted by Dr. Barrett, of Goodna, the blood had been frozen solid. This should be injected intravenously.

The patient is kept up and about, while an ordinary four-hourly chart is kept, for fourteen days, or until the first rigor or rise of temperature. This maintains the patient's strength and lessens the time spent in bed.

At the first sign of an oncoming attack of fever, the temperature, pulse and respiration are recorded hourly, and the patient is placed between blankets and given hot drinks. Blankets *et cetera* are changed as often as necessary. Light diet is given.

The parasites will not be found usually until after the second or even the third rigor. From then on a daily slide should be examined. Leishmann's stain is the best; examination of stained films is more reliable than of unstained blood.

Should no rigor or parasites be observed after twenty-one days, or if only two or three rigors occur, an injection of one cubic centimetre of a one in one thousand adrenalin solution should be given. If the patient harbours a parasite at all, a rigor should occur within four hours. In lieu of this, intravenous injection of an isotonic solution of one gramme of glucose may be tried. Wrapping the patient in hot blankets may induce a rigor. Another injection of malarial blood has in several cases precipitated a rigor. Should no rigor occur

after twenty-five days, successive attempts to infect the patient should be made every seven days.

It is stated, but not observed by us, that infection may occur and persist without fever or rigor. In this case examination of centrifugalized urine and discovery of blood pigment would point to infection.

Splenic puncture may reveal parasites when none are seen in the peripheral blood, and sometimes this operation results in a febrile reaction and the appearance of parasites in the peripheral blood.

If the subcutaneous method of inoculation is used, rigors should occur on alternate days. As a result of intravenous inoculation, a double tertian or quotidian condition soon develops, with a rigor every day; this is undesirable, as it exhausts the patient very quickly.

It should be made quite certain that the infection is pure benign tertian.

Management During the Fever.

General.

The patient should be isolated in a mosquito-proof enclosure, under the observation of an experienced attendant, who should be provided with a list of complications to be watched for.

A rigor, especially the first, is ushered in by restlessness and anxiety; the patient continually wants things, water *et cetera*, for an hour previously. Then the temperature rises and the patient looks distressed and feels hot; after ten to fifteen minutes the temperature drops, the patient shivers, then sweats and becomes more comfortable and settled. He then becomes either drowsy or maniacal. If the latter, he requires sedatives.

The blood pressure is recorded daily and a curve of the blood pressure is kept, the pulse pressure during the paroxysm being always recorded. If the systolic pressure fall below 100 millimetres of mercury, caffeine in a dose of 0·12 gramme (two grains) is given; if below 90, the fever is terminated.

Sponging a patient is contraindicated, as the pyrexia is probably beneficial, and in any case the temperature will fall spontaneously.

Restlessness and delirium are checked by the administration of veronal in a dose of 0·6 gramme (ten grains) as required.

Signs of cardiac distress are treated by ordinary doses of strophanthus as required.

Examination of Blood Slides.

Examination of blood films should be done daily. Leishman's stain should be used. After a few rigors a secondary anæmia becomes evident, numerous microcytes being seen, and the blood platelets increase in number and size, some appearing as definitely organized cell bodies with translucent flagella. In health the average number of platelets per cubic millimetre is 390,000; the number may increase to 500,000 during the course of rigors (the size of the platelets increases at the same time), dropping below 250,000 per cubic milllimetre during convalescence (W. Mackay, *The Quarterly Journal of Medicine*, April, 1931).

In a severe infection, two, three or four plasmodia may be seen in one red cell.

The number of parasites per field should be counted by examining one hundred fields; a sudden increase in the number serves as a signal to cut short the course of rigors.

Complications to be Watched For.

The complications for which a watch should be kept, are set out in the following list.

A. Complications Calling for Termination of the Fever: (i) Hæmaturia; (ii) syncopal attacks; (iii) sudden fall of blood pressure or pulse pressure; (iv) delirium (if the patient's bodily condition is unsatisfactory also); (v) stupor or coma; (vi) epileptiform attacks; (vii) spinal paraplegia; (viii) severe jaundice; (ix) necrosis of phalanges; (x) trigeminal neuralgia; (xi) fall of the systolic blood pressure below 90 millimetres of mercury; (xii) continued fever above 41·1° C. (106° F.); (xiii) sudden rise of the "parasite-erythrocytes" ratio; (xiv) pulmonary or other intercurrent complications of serious nature, such as severe diarrhœa; (xv) sudden decrease in strength, with listlessness and apathy; (xvi) a red cell count of less than 2,000,000 per cubic millimetre; (xvii) increase of pulse rate above 160 per minute; (xviii) increase of respiration rate above 60 per minute; (xix) general paralytic seizures; (xx) increasing amounts of urobilin and urobilinogen in the urine; (xxi) a fast pulse rate, not coming down with the temperature.

B. Complications of Less Importance: (i) *Herpes labialis*, which occurs in over 60% of cases; (ii) mild jaundice; (iii) enlarged spleen; (iv) facial œdema; (v) fall of systolic blood pressure below 100 millimetres of mercury; (vi) headache, which was rarely complained of; (vii) mild diarrhœa; (viii) moderate albuminuria, which occurs in 50% of cases; (ix) mild delirium.

Special attention should be paid to urine tests for bile compounds, as malaria always damages the liver, sometimes fatally, and an infection may be successful and blood destruction may ensue without rigors. This may be discovered by the occurrence of punctate red cells, large mononuclear cells containing pigment, and leucopenia. Even the plasmodium may be seen, and daily urine examination may reveal latent jaundice. Albumin is often found, but not usually in large quantity. Acetone is often found, if the patient is not eating enough. The presence of urobilin and urobilinogen detected in urine diluted to one in four for one day is not dangerous; but if it increases for two or three days the liver damage endangers the patient's life and the course of the disease must be stopped at once.

Test for Urobilinogen (Ehrlich's aldehyde reaction): Take two drops of a 3% solution of paradimethyl-aminobenzaldehyde in a 50% solution of hydrochloric acid, and add to five cubic centimetres of urine. If the reaction is positive, a red colour develops soon, or on warming. Brown is not positive. The above-mentioned salt is very difficult to obtain.

Test for Urobilin (Schlesinger's test): To ten cubic centimetres of urine add one drop of glacial acetic acid and four cubic centimetres of amyl alcohol. Invert several times and allow to stand. Pipette off the upper alcoholic layer and add to it two or three drops of an alcoholic solution of zinc acetate. If a green fluorescence develops, urobilin is present.

Both these tests need not be positive.

Alternative Test for Bilirubin (Latham's test): Drop a few drops of 0·2% watery solution of methylene blue into five cubic centimetres of urine. The appearance of a green colour indicates the presence of bilirubin; the number of drops necessary before the urine turns blue is a rough

indication of the amount of bilirubin present. If more than ten drops are required, the reaction is strongly positive; two to ten drops, weakly positive; if one drop only is required, bilirubin is absent.

Clinical Point to be Noted: The spleen may rupture easily without being much enlarged.

Administration of Quinine.

The following mixture is given in a dose of 14 mils (half a fluid ounce) three times daily:

Quininæ hydrochloridi, 0·3 gramme (five grains)
Aquam chloroformi ad 14 mils (half a fluid ounce)
Solve

One dose of this has been found to clear the system of parasites in one experimental case; but three doses a day should be given for a week, and one dose a day for the following week. One dose of 0·09 gramme (one and a half grains) of quinine bihydrochloride has completely cured several patients of malaria.

When it is necessary to cut short the fever instantly, 0·3 gramme (five grains) of quinine bihydrochloride dissolved in ten cubic centimetres of normal saline solution should be injected intravenously very slowly (two minutes by the clock should be taken over the injection). Any attempt at hurry will produce alarming shock.

Quinine causes some patients to vomit, and it is a good rule never to give more than 0·3 gramme (five grains) at a dose. If vomiting occurs, the dose should be decreased by 0·06 gramme (one grain) at a time, as required, and salol, in coated capsules, in a dose of 0·3 gramme (five grains), should be given with an acid drink with the quinine. If vomiting persists, an intravenous quinine injection should be given in lieu of oral administration.

Instead of quinine "Plasmoquine", in a dose of 0·02 gramme four times a day, may be tried; but in some cases it causes cyanosis due to the formation of methæmoglobin. It should always be given if the malaria turns out to be a malignant form. A dose of 0·12 gramme (two grains) of quinine and 0·03 gramme of "Plasmoquine" daily for a week is often tried successfully in these cases.

Termination of the Fever.

As stated, one dose of 0·3 gramme of quinine hydrochloride may free the patient from parasites, but to make sure, the following procedures should be carried out.

1 A provocative injection of three cubic centimetres of "Phlogetan" is given subcutaneously. A pronounced febrile reaction suggests persistence of the infection.

2. A provocative injection of one cubic centimetre of Parke, Davis and Company's adrenalin solution (one in a thousand) may render parasites visible in the peripheral blood. Blood films are examined hourly for eight hours after the injection, and gametes and schizonts are looked for.

3. Centrifugalized urine is examined for blood pigment, the presence of which points to persistence of infection.

4. Increasing doses of "Neosalvarsan" (0·15, 0·3, 0·45, and 0·6 gramme, given at intervals of five days until 5·5 grammes are taken over a period of fifty days) may be given instead of quinine.

5. To terminate the fever temporarily, 0·12 gramme (two grains) of quinine or 0·15 gramme of "Neosalvarsan" may be given; but in such a case the patient will probably need reinfection from another host (always so in my experience).

After-Treatment.

The figures for the erythrocyte count and the hæmoglobin content fall very greatly, but are quickly restored by the administration of arsenic, as in the following mixture:

Quininæ bihydrochloridi ..... 0·3 gramme (five grains)
Tincturæ ferri perchloridi ..... 0·6 mil (ten minims)
Liquoris strychninæ hydrochloridi .. 0·3 mil (five minims)
Liquoris arsenici hydrochlorici ... 0·3 mil (five minims)
Spiritus aurantii ..... 1·2 mils (twenty minims)
Aquam ad ..... 14 mils (half a fluid ounce)
Signetur: Fourteen mils (half a fluid ounce) to be taken three times a day in water after meals.

In this mixture the quinine and arsenic are combined; it may be used in the second week instead of quinine only.

As soon as the malaria is terminated, the patient is treated as an ordinary convalescent. Supplemental treatment with "Tryparsamide", after convalescence, is usually given.

In my own series of cases (150 in number) I have obtained as good results without subsequent treatment with the arsenobenzols. I gave no arsenobenzol, with the object of proving that the malarial treatment alone produced the improvement. H. R. Unsworth (*The Journal of the American Medical Association*, September 13, 1930, page 772) considers that arsenic is definitely contraindicated in late neuro-syphilis.

Mode of Action of Malaria.

Pyrexia is not now regarded as the important factor, as patients with a normal temperature throughout the course of rigors have improved. It is now believed that the reticulo-endothelial system is stimulated by the malarial toxin to cast off macrophage cells from the endothelium of the blood vessels as independent cells, and the destruction of the treponema is accomplished by phagocytosis exerted by these macrophages, by plasmocytes and by stellate Küpffer cells.

In addition to *dementia paralytica*, malaria therapy has achieved remarkable results in the treatment of interstitial keratitis, hyperplastic bone disease, and malignant nodulo-ulcerative skin lesions of syphilitic origin.

Conclusion.

On April 10, 1927, the present strain of *Plasmodium vivax* was inoculated into a patient who died at Callan Park Mental Hospital last month, after five years as a happy ward worker, and we are now entering the sixth year of its use.

The departmental results to date are as set out in Table I.

TABLE I.

Showing the results of malaria therapy against the results obtained when no treatment was given.

		Percentage.	Percentages for Untreated Patients.
Left hospital able to earn	28	7·0	Nil
Left hospital unable to work	75	19·0	6·6
Greatly improved ..	100	25·0	1·26
Stationary	54	13·5	12·8
Worse	14	3·5	21·6
Died	130	32·0	
Total number treated	401		

At Callan Park no parallelism could be found between the combined physical and mental improvement and the following factors: (*a*) The blood and serological changes; (*b*) the time which had elapsed between the original syphilitic infection and its treatment; (*c*) the time which had elapsed between the first infection and the onset of recognizable general paralysis; (*d*) the type of general paralysis.

Most workers claim to see a greater improvement in patients treated early in the disease, but the statistics in the Callan Park series do not prove this, and I believe that the wish has been father to the thought.

A STUDY OF THE FEET OF NEW-BORN INFANTS.

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EVERY accoucheur must have remarked that the feet of many babies appear deformed and are the subject of anxious inquiry by the mother. In order to investigate the variety of attitudes adopted, the feet of one thousand infants were examined within a few days of birth, most of them within three days, all of them within a week. In the first part of this paper these feet are discussed from the point of view of their resting positions; the second part deals with their range of movement.

THE POSITION OF THE FOOT AT REST.

The method of determining the position at rest was to inspect the foot with the hip and knee each flexed to an angle of 90°. If the infant was asleep, the attitude then adopted was accepted. If the infant was awake, movements were elicited by skin stimuli, and the position to which the foot returned was noted. The symbols used to express the various positions are as follows:

N (neutral) represents the position of the foot when the sole is at right angles to the leg in the sagittal and in the coronal planes. There is no flexion or extension at the ankle, and no inversion or eversion.

C+++ indicates that the dorsum of the foot touches the leg.

C++ indicates that the foot is dorsiflexed more than 45°, but less than *C*+++.

C+ indicates that the foot is dorsiflexed, but less than 45°.

E+++ denotes that the sole of the foot faces directly posteriorly.

E++ denotes that there is more than 45° of plantar-flexion, but less than *E*+++.

E+ denotes less than 45° of plantar flexion.

Varus+++ denotes that the sole of the foot faces directly medially.

Varus++ denotes that the sole faces more than 45° medially, but less than *Varus*+++.

Varus+ denotes that the sole faces medially less than 45°.

Valgus+++ indicates 90° of eversion.

Valgus++ indicates between 45° and 90° of eversion.

Valgus+ indicates less than 45° of eversion.

While it was no object of the investigation to inquire into the occurrence of talipes (a study of two thousand unselected feet could throw no light on the statistics already existing), a look-out was nevertheless kept for examples of definite deformity.

The first fact to emerge is that the neutral position is by far the commonest attitude for the foot of the new-born infant at rest. In this series, 1,192 feet (59·6% of the feet examined) were in the neutral position. The attitudes next in order of frequency were: Calcaneus, 17·3%; varus, 13·2%; calcaneo-varus, 6·55%. The remaining attitudes, calcaneo-valgus, equino-varus, equinus and valgus, are uncommon and account for less than 3% between them (see Table I).

TABLE I.

Showing the Position of the Feet of Newly Born Infants at Rest.[1]

Position at Rest.	Number of Feet.	Percentage in each Position.
Neutral	1,192	59·6
Calcaneus	346	17·3
Varus	264	13·2
Calcaneo-varus	131	6·55
Calcaneo-valgus	27	1·35
Equino-varus	17	0·85
Equinus	12	0·6
Valgus	11	0·55
Equino-valgus	0	—

[1] In this table only deviation at the ankle and subastragaloid joints is considered. In eighteen cases *metatarsus varus* was present, but these are discussed in a later paragraph.

Paired Feet.

The right and left feet of 819 infants (81·9%) were in the same position, whether neutral or otherwise. Also, of ten infants encountered with *metatarsus varus*, eight were affected in both feet. Two of the three examples of *talipes equino-varus* were in the same infant.

RELATIVE FREQUENCY OF VARIOUS FORMS OF CONGENITAL TALIPES AND VARIOUS RESTING POSITIONS.

Any statistics of congenital talipes will show that equino-varus is by far the commonest deformity and calcaneus one of the rarest. Of the attitudes adopted by the infant foot, however (excepting those in the neutral position), calcaneus is the commonest, 42·82% (346 examples of 808 not in the neutral position), and those in equino-varus number only 17 (2·1%), including three examples of fixed deformity.

The complete figures for this series, compared with the figures for talipes quoted by Whitman, are

shown in Table II, but in passing there are two conclusions which may be drawn.

In the first place it is obvious that although 40·4% of infants' feet at birth adopt attitudes which, if persisted in until adult life, would be deformities, the overwhelming majority of them are not even potential deformities, but fall into line with the adult normal, probably coincidently with the gradual adoption of normal function.

Secondly, if the attitude at birth is a persistence of the attitude *in utero*, and I feel from watching a number of confinements that it is so, then these figures throw doubt on the theory that most cases of talipes have the same cause. Whitman ("Orthopædic Surgery", eighth edition, page 827), discussing the ætiology of congenital foot deformities, says: "The most reasonable explanation as applied to the majority of cases is the mechanical. This in brief is the theory that the foot has for some cause remained for a longer or shorter time in a constrained or fixed position and has thus grown into deformity." He quotes Bessel-Hagen in support of his view. Others, of course, do not hold this view, but maintain that congenital deformities are due to an arrest of development. The present figures support this latter theory. Another very striking example, where a *talipes varus* becomes a *talipes equino-varus* after birth, is mentioned below.

TABLE II.

Comparison between the relative Frequency of the Various Forms of Congenital Talipes, and the relative Frequency of the Various Positions at Rest in this Series.

Attitude.	Talipes. (after Whitman.)	Resting Position in this Series (excluding numbers in Neutral Position).	
	Percentage of Total.	Number.	Percentage of Total.
Equino-varus	77·4	17	2·10
Valgus	6·8	11	1·35
Varus	4·2	264	32·67
Calcaneo-valgus	4·1	27	3·34
Equinus	2·3	12	1·48
Calcaneus	2·2	346	42·82
Equino-valgus	1·6	0	0
Calcaneo-varus	0·47	131	16·21
Total	2,103	808	

In addition, I found 18 examples of *metatarsus varus*, or 0·9% of all feet examined. The figures quoted by Whitman do not include any cases of this deformity, though in his text book he does give a short paragraph setting out a definition and proposals for treatment.

Metatarsus Varus.

I found 18 examples of *metatarsus varus* among 1,000 new-born infants. This figure is significant from several points of view. In the first place the condition is given no mention in some standard text books, and I could find only one reference to the subject in the "Index Medicus" for the past ten years. That it is not as transitory a condition as the other positions at rest discussed above is obvious from the figures of the out-patients' department at Saint Vincent's Hospital, where, although adult patients greatly predominate, there is at least one child brought every year for treatment of *metatarsus varus*. That it does tend towards spontaneous recovery, however, is also obvious, as the proportion is much less than eighteen per thousand, and also because it is apparently rarer still as a separate deformity among adults.

As medial deviation of the forefoot is a constituent part of all cases of *talipes equino-varus*, I investigated the eighteen cases in this series to see if there was any tendency towards that deformity. To do this, I considered the position at rest of each foot affected with *metatarsus varus*, to see if there was a preponderance of equino-varus attitudes, and compared the range of movement in each with the range (or lack of it) in club-foot. The results are shown in Tables III and IV.

TABLE III.

Position at Rest associated with 18 cases of Metatarsus Varus.

Position at Rest.	Number of Cases.
C+Varus+ ..	8
N	6
Varus+ ..	2
Varus++ ..	1
E+Varus+++	1
Total	18

TABLE IV.

Range of Movement associated with 18 cases of Metatarsus Varus.

Position at Rest.	Range of Movement.			
	Dorsi-flexion.	Plantar-flexion.	Inversion.	Eversion.
1 *C+Varus+*	+++	+	+++	++
2 *C+Varus+*	+++	+	+++	++
3 *C+Varus+*	+++	++	++++	++
4 *C+Varus+*	+++	++	+++	++
5 *C+Varus+*	+++	++	+++	++
6 *C+Varus+*	+++	++	+++	++
7 *C+Varus+*	+++	++	+++	++
8 *C+Varus+*	+++	++	+++	++
9 *N*	+++	++	+++	+
10 *N*	+++	++	+++	+
11 *N*	+++	++	+++	++
12 *N*	+++	++	+++	++
13 *N*	+++	++	+++	++
14 *N*	+++	++	+++	++
15 *Varus+*	+++	++	+++	++
16 *Varus+*	+++	++	+++	++
17 **Varus++*	+++	++	+++	0
18 **E+Varus+++* ..	+	++	++++	+

It will be seen that, judged by the attitude of the foot, in 17 out of the 18 cases there was no tendency towards *talipes equino-varus*. This is what one would expect. If an examination of 2,000 feet revealed 18 with a tendency towards club-foot, one would anticipate a figure in excess of that stated by Jones and Lovett ("Orthopædic Surgery", second edition, page 614), namely, that "equino-varus occurs about once in every one thousand births".

However, in two of the cases (marked with an asterisk) there is a resemblance to *equino-varus*,

judged by their respective range of movement, though in one of these the tendency is much less marked than in the other. The latter, number 18, was examined at the age of twelve months, and the results are tabulated later. It is sufficient to state here that the *metatarsus varus* condition had disappeared. The other example (number 17) could not be traced.

Fixed Deformities.

Three examples of *talipes equino-varus* and one of *talipes varus* were seen in 1,000 unselected new-born infants. This latter deformity, examined one year later, disclosed the fact that it is now a *talipes equino-varus* (see below).

Range of Movement.

To determine the range of movement, the foot was manipulated into each of the four primary directions, gentle force only being used. The symbols representing the range in each direction are the same as those used to indicate the position at rest and have the same meaning. In addition, *C O*, *E O*, *Varus O*, and *Valgus O* are used to show absence of movement in the direction of dorsiflexion, plantar flexion, inversion and eversion respectively.

Table V shows the number of feet which could be manipulated into each of the various positions.

TABLE V.

The Range of Movement of the Feet of 1,000 New-Born Infants.

Movement.	Number.
C+++	1,885
C++	112
C+	3
C O	0
E+++	21
E++	1,844
E+	129
E O	6
Varus+++	1,947
Varus++	53
Varus+	0
Varus O	0
Valgus+++	22
Valgus++	1,704
Valgus+	269
Valgus O	5

This table shows how much greater is the range of movement of the infant foot compared with that of the adult, though the difference is relatively more marked in dorsiflexion and inversion than in plantar flexion and eversion. For example, whereas the commonest range in the new-born infant can be represented by *C*+++, *E*++, *Varus*+++ and *Valgus*++, the adult range would have to be represented by *C*+, *E*+ to ++, *Varus*+ and *Valgus*+.

It is difficult to assess the relative parts played by skeletal and non-skeletal structures in permitting this wide range of movement in the infant, but lack of muscle tone is an important, though not the sole cause. For instance, tenotomy of the *tendo Achillis* of an adult, while permitting an increased range of dorsiflexion, does not allow the dorsum of the foot to be brought into contact with the front of the leg, which *C*+++ denotes. However, tension of the other soft parts behind the ankle, including the deep calf muscles and the skin (an important factor), has to be considered. Similarly, inversion in the adult is largely checked by the tension of the peroneal tendons, as can be shown by tenotomy or paresis of these muscles. Probably the shape of the skeleton has least to do with the difference in range, and muscle tone the most. This view is supported also by those cases in this series in which the range was less than the average. Where, for example, the foot could be manipulated into dorsiflexion only to a degree indicated by *C*+, I felt that the resistance to further movement was elastic and due to muscle tone and soft tissue tension, and not to skeletal impingement.

The numbers in Table V for the range in certain directions are so small that it is justifiable to claim that *C*+, *C O*, *E O*, *Varus*+, and *Valgus O* denote a pathological degree of deficient movement, and that feet affected by these deficiencies may be grouped together for discussion.

Deficient Movement.

On reference to Table V it will be seen that there are fourteen examples of deficient movement. The four examples of fixed deformity account for five of them; the remaining nine represent one foot each. I succeeded in tracing most of these cases about a year afterwards. The results are discussed in a later paragraph.

Three feet were deficient in the movement of dorsiflexion. At rest two were in the position of equino-varus, one in varus. One of these (its attitude at rest was *E*+ *Varus*+++) I have included in the fixed equino-varus deformities, because it was also in a *metatarsus varus* attitude. Another (*Varus*++) was deficient in eversion as well as dorsiflexion; it could not be manipulated into any degree of eversion. This foot is classed as *talipes varus*. Investigation of these two feet a year later discloses very unexpected changes, which will be mentioned later. The other foot (*E*+ *Varus*+ attitude at rest) was otherwise not remarkable.

Five feet were deficient in the movement of eversion. One of them (*talipes varus*) is discussed in the previous group, as it was also defective in dorsiflexion. Another (its position at rest was *Varus*++) was also in a *metatarsus varus* position. It resembled club-foot in three important particulars, varus, *metatarsus varus* and deficient eversion. The after-history of this case would also be interesting; but I have been unable to trace it. Perhaps it should be called tentatively *talipes equino-varus*, and included among the fixed deformities. I did not so include it, because the foot did not have the unyielding feel characteristic of feet in that group.

The third example (*E*+ *Varus*+ at rest) seems to represent a still earlier stage or slighter degree of deformity.

The remaining two are very definite club-feet.

Six feet were defective in their degree of plantar flexion. They were all in the calcaneus position at

rest, and one of them had a valgoid tendency as well. Apparently the structures in front of the ankle were shortened; but in view of the rarity of *talipes calcaneus*, the presumption is that function will restore these feet to the adult normal. Later examination confirms this.

In Table VI the findings in this group are summarized. It will be noticed that no foot in the *N* position at rest had deficient movement.

TABLE VI.
Various Deficiency in the Range of Movement, and the Resting Positions of the Affected Feet.

Deficiency of Movement.	Number of Examples.	Position at Rest.
C+	3	*E+Varus+++* (*talipes equino-varus, metatarsus varus*). *Varus++* (*talipes varus*, defective eversion). *E+Varus+*.
C O	0	
E O	6	*C+*. *C++* (4). *C+Valgus+*.
Varus+	0	
Varus O	0	
Valgus O ..	5	*Varus++* (*talipes varus*, defective dorsiflexion). *Varus++* (*metatarsus varus*). *E+Varus+*. *Talipes equino-varus* (2).

NOTE: For explanation of the symbols see text. Associated deformities are shown in parentheses.

After-History.

I was able to trace seven of the cases in which there was deficient movement. Five of the patients at birth had deficient plantar flexion and a calcaneus attitude of the foot. In periods varying from eleven to fourteen months after birth, examination showed that the defective movement had disappeared, and at rest each foot was in a position of slight equinus, as is the adult foot. None of them had been subjected to treatment.

The remaining two feet are very interesting, and demonstrate opposite tendencies. These at birth are indicated in the following parallel statement.

Position at Rest.	*Metatarsus Varus.*	Range of Movement.
E+ Varus++	Present	*C+ E+ Varus++++ Valgus+*
Varus++	Absent	*C+ E+++ Varus+++ Valgus O*

The first, which was included in my list of fixed deformities, has become a normal foot without treatment. The equino-varus and *metatarsus varus* tendencies have disappeared, the range of dorsiflexion has greatly increased. The excessive inversion has vanished. The second has become a definite club-foot, retaining the same deficiency of movement; medial deviation of the forefoot typical of such cases has been superadded.

SUMMARY.

1. The feet on one thousand new-born infants were examined.
2. Of the feet examined 40·4% occupied, at rest, positions which would be deformities if they persisted.
3. Eighteen examples of *metatarsus varus* were seen.
4. Four other examples of deformity were seen—three of *talipes equino-varus* and one of *talipes varus*.
5. The relative frequency of the various positions at rest in this series bears no relation to the relative frequency of the various forms of congenital talipes.
6. This investigation supports the "endogenous disturbance" theory of the ætiology of congenital foot deformities.
7. The developmental imbalance which causes club-foot can continue after birth.
8. Feet which possess a fixed deformity at birth can, without treatment, become normal after birth.
9. The infant's foot is capable of much greater range of movement than that of the adult.
10. Feet possessing a deficient range of movement at birth can acquire the normal range after birth.

ACKNOWLEDGEMENT.

I take this opportunity to thank the members of the honorary staff, the Medical Superintendent, the Matron and the nurses of the Royal Hospital for Women, for making the babies under their care available to me. Also, I must gratefully acknowledge the help and criticism of Dr. D. J. Glissan, which were always readily forthcoming.

Reports of Cases.

AMENORRHŒA WITH RESPONSE TO HORMONE THERAPY.

By H. DARBY THOMAS, M.R.C.S. (England), L.R.C.P. (London),
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V.C., AGED twenty-one years, was first seen by me on March 7, 1930. She complained of amenorrhœa of eighteen months' duration and increase in weight from her normal 59·4 kilograms (nine stone six pounds) to 73·8 kilograms (eleven stone ten pounds). These symptoms followed a severe attack of rheumatic fever, for which she had been laid up four and a half months. Her menstrual periods had previously been regular, every twenty-eight days, and had lasted five days. Before I saw her she had been treated by "gland tablets" and had had "an operation on her womb", with no effect. She had had four attacks of rheumatic fever and had had scarlet fever and diphtheria in childhood.

She was a stout girl of normal secondary sexual developments and good colour. The uterus was anteverted and of normal size. On March 20 she began taking "Agomensin (Ciba)", four tablets a day. On March 27 normal menstrual period began and lasted five days. For the next two months she took "Agomensin" before each period was due, and menstruation continued regularly

until December. She lost over 6·3 kilograms (one stone) in weight, felt well, and resumed her household duties.

The December menstrual period was missed; menstruation in January, 1931, was normal, and in February there was only a slight loss, lasting two days; the March period was missed. She resumed "Agomensin" on April 6 and took the tablets for the next two or three months. Menstruation continued regularly every twenty-eight days until October, when the loss was small. She did not menstruate in November or December. I then gave her intramuscular injections of "Agomensin" every four days; after the fifth injection a normal menstrual period appeared.

During each period of amenorrhœa she gained excessively in weight, but lost it, without change of diet, whilst menstruating, and felt much better and more active. The response to treatment was so pronounced on each occasion that it must be attributed to an active ovarian hormone present in "Agomensin".

Reviews.

THE PROGRESS OF HYGIENE.

THOSE of us who are accustomed to talk of the "good old times" should read Dr. Anthony Delmege's book on the history of national health.

The author has traced in full and interesting detail the gradual evolution of the idea that the health of the people is the concern of those in authority and that ill health and lack of sanitation do not pay.

It has taken centuries of effort on the part of many devoted men and women to persuade the average citizen that it is a dirty and unhealthy habit to throw refuse into the street, and the process of education has not yet been completed. Up to the beginning of the last century conditions were appalling, and the modern medical practitioner can only look back with wonder and admiration at the powers of resistance to disease developed by our great grandfathers. They drank water from wells which were cesspits, their houses had little or no ventilation, and the conditions under which their food was slaughtered and kept are incredible to modern readers. In spite of it all, many of them lived to an advanced age.

It is a very sad and depressing fact that mankind progresses up to a certain point and then the progress and advances are lost. Later generations go all over the same ground. In the civilization of Crete, which flourished from 3000 to 1800 B.C., there was the first known example of a scientific system of sewage disposal, as recently discovered in excavations at Knossos. Earthernware pipes, cement conduits, primitive water closets and a water flushing system. Bathrooms, sedimentation tanks and all the other adjuncts of modern sanitation are to be found. In England in 1800, roughly four thousand years later, the sanitary conditions in the large and small towns were hopelessly primitive and filthy. All the lessons and advances of Crete, Egypt and Rome had been completely forgotten, to be painfully relearnt in the later years of the last century.

During the dark ages the light of science died down to the merest glimmer in Europe, and it is to the Arabs that any progress in public hygiene is owed. The great cities of Alexandria and Damascus in the East, Cordova and Seville in Mohammedan Spain, all rejoiced in well-equipped hospitals and healthful sanitary regulations. Throughout this period in Europe learning was kept alive in the monasteries; but alas! the monastic idea of private hygiene was primitive in the extreme. In the Benedictine monastery of Monte Cassino, founded in A.D. 525, the following rule was in force: "Let the use of baths be granted to the sick as often as it shall be expedient, but to those who are well, and especially to the young, baths shall seldom be permitted." Christianity, while it did much for the care of the sick and indigent poor, did little for private or public hygiene and cleanliness.

A most interesting, if appalling, description of life in mediæval England is given. Streets were narrow and unpaved; animals were slaughtered in the streets, vermin swarmed on the persons of rich and poor, and in the houses and inns. "*Pulex, culex, cimex*" was proverbial in mediæval times, and the frequent fires must have been a godsend.

The great plagues and epidemics are described, and how, slowly, their virulence was sapped by improved conditions. The control of leprosy, the great prevalence of occupational diseases—lead and mercury poisoning were exceedingly common—all these are dealt with in pages full of interest.

Conditions in the "spacious times of good Queen Bess" showed very little improvement. The Queen evidently made some effort to set a better example to her subjects, for we find one author expressing his surprise that the Queen "doth bathe herself once a month whether she requires it or not". Soap was a luxury, and most of the people used substitutes, such as wood ashes, hemlock, nettles and cow-dung.

The eighteenth century was to see the growth of a school of medical hygienists who, appreciating the necessity of changes in conditions of living for the sake of the people's health, strongly advocated reforms. Such men were Mead, Percival, Ferriar of Manchester, Currie of Liverpool, and Haygarth of Chester. Howard, the philanthropist, travelled all over England and Europe and brought about a long-needed reform in the treatment of prisoners and lunatics. Pringle in the Army, and Lind in the Navy, reformed the health conditions of the sister services.

The second decennium of last century found a state of affairs in England very similar to that of the present time. The Napoleonic wars were over, a period of deflation and business depression followed, and all schemes of hygienic reform lapsed. Food prices were high, and many people starved. Strange to say, the health of the population improved, and a "general decrease of disease occurred during a prolonged period of national distress". Then in 1832 conditions gradually improved, farming took advantage of the scientific discoveries that were being made, and food became plentiful and cheaper. England had begun to increase her manufactures at the end of the eighteenth century and now entered fully an era of manufacturing prosperity. The rapid increase of population and its concentration in certain centres led to new problems in public health; child labour, housing, regulation of factory workers and many other problems had to be solved. These problems were taken in hand by such men as Robert Owen, Lord Shaftesbury, Southwood Smith, and that highly unpopular reformer, Edwin Chadwick, who, as our author says, "was never able to cultivate the *suaviter in modo*". Space only permits us to mention John Simon, Chadwick's successor, who became one of the earliest medical officers of health; Florence Nightingale, whose name is a household word in the nursing profession, was also one of the first experts in sanitary reform.

The whole story is a fascinating, if sad, one. The reader shudders at the cruelty with which children were treated, even by their own parents; at the horrors of the asylums, prisons, factories and mines; the superstition, greed and lack of human feelings which blocked the path of progress during the past centuries; but he is thrilled by reading of the noble and self-sacrificing efforts of such men as Howard and Chadwick, and proud to belong to the same profession as Percival, Ferriar and Lind.

The book is enriched by numerous interesting illustrations and portraits. It should be read by all members of the medical profession and by all those interested in the history of the efforts to improve the health of the people.

[1] "Towards National Health, or Health and Hygiene in England from Roman to Victorian Times", by J. A. Delmege, O.B.E., M.R.C.S., L.R.C.P., D.P.H., with a foreword by Sir Thomas Legge; 1931. London: William Heinemann (Medical Books). Crown 4to., pp. 248, with illustrations. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, JUNE 25, 1932.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

HUGHLINGS JACKSON.

TWENTY-FIVE years ago, in the National Hospital for the Paralysed and Epileptic, there was placed a marble bust of John Hughlings Jackson as a mark of honour and esteem for one of its greatest consultants, a man who, in the words of Sir James Paget, "had given lucidity to physiology and guidance to surgery". A further and much needed memorial to this man's memory has just been completed—two large volumes of his "Selected Writings", edited by James Taylor in association with Gordon Holmes and F. M. R. Walshe. Here for the first time are collected those many contributions on convulsions, cortical localization and cerebral evolution and dissolution, which, delivered as lectures or scattered through the periodical medical literature of the times, have become embodied in the modified neurological teaching of today.

The fertile mind of Hughlings Jackson ranged over the entire province of neurology. He envisaged the unity of the organism, and his mind went deep to the anatomical structure and pathological change which lay beneath disordered clinical functions. His addresses were numerous, his writings prolific. A thinker with a strong philosophic bias, an investigator with a sense of logic, he was ever the teacher, ever the expounder of his theories. He wrote generously and brilliantly to the medical press. In the thirty odd years of his medical maturity his published contributions numbered over two hundred; and in three consecutive years no fewer than forty neurological papers emanated from his pen. Apparently he was too busy to write a book, although when asked by the late William Osler, he once promised to collect, edit and arrange his contributions to neurology. That was thirty years ago; and although he lived on and worked for another ten years, he never prepared the manuscript. It has now been done for him by Taylor and his associates, who cooperated in doing what, although an extremely arduous task, was none the less a labour of love.

These two handsome and well arranged volumes not only serve to commemorate the life works of a great clinician, a keen observer, a prodigious worker and a pioneer in what was then the largely unexplored realm of neurology, but they bring the reader into actual contact with the basic principles of cerebral structure and function upon which much of our present day knowledge of neuro-psychiatric mechanisms has been built by later hands. They do more. They stimulate a fresh interest in the old and still unsolved problems by reason of the original and often provocative views expressed in the writings of this brilliant yet modest teacher. Hughlings Jackson was early associated with the study of aphasia and with the ophthalmological aspects of cerebral tumour and intracranial pressure. He cast his searchlight into the problems of neurosyphilis. He made contributions on the subject of hemiplegia. He peered into the mysterious depths of dementia and cerebral dissolution. He amplified the prevailing views on vertigo. He gave new orientation to the knowledge of cerebral function and the inhibitory action of the higher levels. But, above all this, there was one subject which he made particularly his own. The name of Hughlings Jackson is synonymous with epilepsy wherever neurology is taught. He studied every aspect of the convulsions, from the gross discharge of *grand mal* to the isolated myoclonic twitchings of focal origin and the subtle sensory changes in the

so-called uncinate fit. His "Study of Convulsions" (1870) and his Lumleian Lectures on "Convulsive Seizures" (1890) are monuments of philosophic insight and clinical acumen.

Though to turn back from a perusal of the modern biochemical and hydrodynamic research on the subject of epilepsy to the writings of Hughlings Jackson may be like going from the roof garden of a building to the cellar, the experience is most salutary, for here we see how strong and adequate and unyielding are those foundations which were set by the sure and masterly hand of Hughlings Jackson.

Some of his theories have been discarded; many of his ideas have been modified; but when one considers just what kind of a medical wilderness neurology was when Hughlings Jackson embarked upon his life work, the wonder is not only that he went so far, but that he made so few mistakes. His work still bears fruit and will continue to do so. The men who came into personal contact with him and those who number themselves proudly among his students are passing away. But some of these have fortunately seen fit now to honour and perpetuate the name of him who so becomingly adorned his profession; and these volumes of his "Selected Writings" will long remain an inspiration to those who seek to continue the trail he blazed.

Current Comment.

RHEUMATIC LUNG LESIONS.

THE occurrence of abnormal physical signs in the lungs of patients seriously ill with rheumatic fever has been recognized for many years. The pathological states suggested to the clinician were pleurisy with or without effusion, lobar pneumonia and collapse of the lung due to the pressure of a grossly enlarged heart or pericardial sac. Accurate knowledge has been slow in coming, for patients suffering from acute rheumatism rarely die in the first attack, and the long standing cases do not present the best pathological material for such a study, owing to the complication introduced by the passive congestion in the lungs incident to cardiac failure. A valuable contribution was made by B. A. Gouley and J. Eiman to this subject some five years ago, when they published histological studies of the rheumatic lung; the same authors have now brought forward an interesting series of nine cases, with full histories and autopsy reports.[1] Their work is along lines similar to that reported by A. E. Naish in England a few years ago, and their observations help to provide that necessary firm basis of pathology on which alone is it possible to build an understanding of disease.

Gouley and Eiman point out that the physical signs are much more prominent in the lungs than are the clinical symptoms referable to them. Dulness to percussion is found, and bronchial breath sounds are heard; moist adventitious sounds may be heard, but not so frequently as in other types of pneumonic consolidation. Such a consolidation is at times transient only, but on the other hand, may last for many weeks. It may be accompanied by involvement of the pleura, sometimes with effusion. The patients with the more severe types of carditis are those most commonly affected; but it is of interest that there is not of necessity that exacerbation of symptoms that might be expected with a lobular or lobar pneumonia of the usual kind. Nor can a simple atelectasis, from an increased bulk of the cardiac apparatus, be the cause, since the lesions may be scattered and appear in the right lung as well as in the left. Thus it will be seen that there is a good *a priori* case for a specific lung lesion. Does this recent work support previous claims for such specificity on purely anatomical grounds? The answer is definitely yes.

In all the cases studied by Gouley and Eiman the illness was acute; that is, the patients died in the acute stage of the disease, and presented clear pulmonary signs during life. The consolidations were less bulky than those of lobar pneumonia, and the cut surface of the affected part was red, firm, finely granular, and moist. The appearance has been compared to solid red india-rubber, and the descriptive name of "splenization" has been used. Microscopically, there was perivascular infiltration, interstitial in distribution, and the alveolar walls were thickened by outgrowths of new cells. The characteristic feature of the exudate was the presence of large endothelioid cells identical morphologically with those found in rheumatic lesions of the heart. It is pointed out by the writers that some doubt has been cast on the real specificity of these changes. Since Aschoff described the rheumatic nodule, however, very few similar conditions have been found. *Streptococcus viridans* pneumonic lesions, especially in the laboratory animal, and the pulmonary inflammatory foci in subacute bacterial endocarditis also produce an endothelioid type of reaction; nevertheless, the changes found by Gouley and Eiman are so characteristically those of other acute and subacute rheumatic lesions that their true nature can hardly be doubted.

The really interesting point about this work is that the patients studied were affected with pneumonic consolidations complicating acute rheumatism. Those with pleuritic changes only were excluded, for these changes are much more common and generally better known. The atelectases associ-

[1] *The American Journal of the Medical Sciences*, March, 1932.

ated with pressure on the lung were likewise omitted. Therefore, we have still further convincing evidence that rheumatic consolidation of the lung is a clinical and pathological entity. The older clinicians wrote about this a good many years ago, but the absence of histological proof was responsible for a scepticism that caused the diagnosis of such a condition to be forgotten at the bedside. There is no longer an excuse for this. Fortunately the presence of a rheumatic pneumonitis (which is the most accurate descriptive term) does not *per se* make the prognosis more serious, but it occurs in the more severe infections, and is in this way of some prognostic importance.

Acute rheumatic fever is not so very common in this country; but it is seen too frequently for our peace of mind, and any piece of knowledge that augments the understanding of a malady so damaging, so prolonged as regards the period of treatment required, and so largely handled by the general practitioner, must be of the highest interest and value.

INTRACRANIAL ANEURYSMS.

IT is a truism that no accretion of scientific knowledge can ever be said to be purely of academic interest; at least it should be remembered that those who rashly dismiss any work as lacking in practical application frequently have to eat their words. The old-fashioned morbid anatomist has often been the butt for criticism in the last few decades, yet his observations are still full of suggestions to the clinician.

It may be thought that intracranial aneurysms give sport only to the neurologist and the *post mortem* expert; but, as knowledge accumulates with regard to the congenital nature of many of these anomalies, also the surprising length of time that may elapse before rupture occurs, and, more important still to the practising physician, as the recognition of "spontaneous" subarachnoid hæmorrhage becomes more general, it will be seen that the subject actually concerns the pathology of the living.

W. H. Chase has recently described in detail an extraordinary case of intracerebral aneurysm.[1] The patient was a woman, aged thirty years, who was eight months advanced in pregnancy. She was found lying unconscious on the floor. Examination revealed a state of coma. She responded reflexly to painful stimulation; the limbs were flaccid, but Kernig's sign was present on both sides. The urine was of normal specific gravity, and contained sugar and ketones, but no protein. As after a few hours she became cyanosed, Cæsarean section was performed successfully, and a living child was delivered. The mother died immediately afterwards. The tentative clinical diagnosis was eclampsia or possibly cerebral tumour. Lumbar puncture was not performed. At autopsy a single large sacculated ruptured aneurysm of the right middle cerebral artery at the site of its cortical branches was found. It was entirely within the cerebral hemisphere. A remarkable feature was the huge size of the aneurysm; it is amazing that such great dimensions could be attained without previous warning. Histological examination proved that there were no signs of a granulomatous or degenerative process, such as syphilis, in the vascular system. This is important, for, though it is well known that aneurysms of the circle of Willis are found in cerebral vascular syphilis, it is quite erroneous to assume that all such are due to this cause. Chase demonstrated in this case that there were defects in the *tunica media* of the artery at the points of branching, and also that both posterior communicating branches of the circle of Willis were congenitally absent. There were also anomalies in the anterior chorioidal arteries.

In a paper read at the Third Session of the Australasian Medical Congress in 1929, P. P. Lynch reported several cases and laid stress on the frequency with which other sacs were found in addition to the ruptured one, and also the presence of congenital variations and malarrangements of the vessels, especially the circle of Willis.

Now recognizing the not very uncommon occurrence of intracranial aneurysms, and admitting that these are frequently of congenital origin, what is the importance of this knowledge? First, as Chase has demonstrated in this case, and as others have shown, the essential defect is not necessarily the presence of an aneurysmal sac *per se*, but a weakness of certain parts of the arterial wall, particularly in the middle coat, and in the vicinity of branches. This explains why patients may lead active and previously healthy lives over long periods of time, in some instances well over the thirty years attained by this woman, before the serious and often fatal accident of rupture occurs. Secondly, the clinical syndrome of subarachnoid hæmorrhage should be realized as one of importance. The accident of rupture does not always cause death, but it produces a train of symptoms indicating severe meningeal irritation, sometimes even simulating acute meningitis. It will be noted that in Chase's case Kernig's sign could be elicited on either side. The patients may pass sugar and ketones in the urine; some have actually been treated for supposed diabetic coma. The diagnosis is, however, readily made when bright and often apparently pure blood is obtained by lumbar puncture. It will be seen that when such a patient recovers, and this may happen unexpectedly sometimes, an extremely doubtful prognosis must be given, for there is no certainty that the aneurysm will not leak again; and, absorbing the lessons of morbid anatomy, the clinician must remember that where there is one congenital lesion there may be others, so that the cycle of events may quite easily be repeated in the same place or in another.

[1] *The Journal of Pathology and Bacteriology*, January, 1932.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, Albert Street, East Melbourne, on October 7, 1931, DR. VICTOR HURLEY, the President, in the chair.

Toxic Goître.

DR. R. J. WRIGHT SMITH read a paper entitled: "Toxic Goître."

In introducing his subject, Dr. Wright Smith remarked that the pathology of toxic goître presented a problem that was still far from solution. A correct interpretation of the pathological changes was impossible without a consideration of the development, structure and function of the thyreoid gland. He then discussed briefly the histology of the thyreoid gland.

He remarked that it had been customary to divide toxic goître into two or more types; for example, toxic adenoma and exophthalmic goître. This division had been made mainly on clinical grounds. It was doubtful, however, if any such distinction was possible. Many observers had noted that the response to iodine was essentially the same, whatever the clinical type of goître. Virchow had believed that all goîtres were essentially the same, differing only in their mode of development.

A phenomenon common to all forms of goître was epithelial hyperplasia. While adenoma of the thyreoid gland did occasionally occur, the term "toxic adenoma" was misleading, and, in most instances, was without pathological justification. When an adenoma was present in a gland that was producing symptoms of thyreotoxicosis, the cause of the hyperthyreoidism should be sought, not in the growth, but in the surrounding glandular structure.

Dr. Wright Smith proceeded to discuss the histology of toxic goître. He dealt with the hyperplastic and involutionary changes that occur, and showed how large areas of colloid material could become surrounded by fibrous tissue and thus present the appearances to which the terms "colloid cyst" and "colloid adenoma" are sometimes applied. He referred to Rienhoff's observations on the hypo-involutionary and hyperinvolutionary changes that take place as a result of the administration of iodine to persons suffering from goître. He remarked that it could be understood how hyperplasia and subsequent involution and repetition of this process could cause a thyreoid gland to become nodular and thus to suggest adenoma.

He drew attention to the similarity between the histological appearances of carcinoma and toxic goître, and remarked that in some cases there appeared to be a definite transition from thyreoid hyperplasia to carcinoma. Carcinoma did not usually develop in a gland that was not goîtrous. In some instances the histological appearances of colloid goître, epithelial hyperplasia and carcinoma could be observed in the one gland.

Dr. Wright Smith mentioned the diversity of opinion concerning the association of toxic goître and endemic goître, and remarked that the difference between these two types was in degree rather than kind. He concluded with the remark that it was difficult to bridge the gap between the physiological hyperplasia of a colloid goître and the pathological hyperplasia of a toxic goître.

DR. JULIAN SMITH read a paper entitled: "Toxic Goître" (see page 877).

DR. S. V. SEWELL, in discussing the medical aspect of toxic goître, said that interest in the goître problem had been largely stimulated in Victoria by the pioneering work of Dunhill. Brilliant work in the management of those catastrophic cases of neglected toxic goîtres had made his name justly famous throughout the world. However, his interest had lain mainly in devising means to overcome the technical difficulties of operation at a time when chloroform and ether given by the closed method were almost the only anæsthetics in use. He had achieved success in hosts of well-nigh hopeless cases, in which there was grave circulatory failure, by operating under local anæsthesia. Such hopeless cases were not seen in these enlightened days, and it was impossible for younger medical practitioners to visualize the parlous condition of the patients whose treatment Dunhill tackled so bravely. Speaking of the classification of toxic goître, Dr. Sewell said that it was impossible to make a classification on an ætiological basis, as they were still far from understanding the causal factors. For the present he was prepared to accept the working classification of Graves's disease and nodular toxic goître, because it was simple and useful. Graves's disease was a disease of more or less acute onset in which the whole thyreoid tissue became hypertrophic and hyperplastic. The symptoms of this condition might be severe or mild, and in the severe case the diagnosis was so obvious that in these days mistakes were not made. However, it had to be recognized that many of the mild cases were overlooked, and in some cases diagnosis was not by any means easy, since exophthalmos and an obvious goître might both be absent. Such patients often presented themselves complaining of loss of weight and strength, general nervousness, irritability, restlessness, and were frequently looked upon as suffering from neurasthenia. The correct diagnosis could usually be made without special tests, as there was usually a characteristic stare, due to the overaction of the sympathetic supply to the involuntary musculature of the upper eyelid, which was thus slightly raised, so that the upper limits of the iris were exposed instead of the eyelid, as in normal people, coming down half way between the upper limit of the iris and the pupil. This characteristic stare was not present in neurasthenic patients. Tachycardia was also almost always present. Another important diagnostic finding was the increased surface warmth, both subjective and objective, due to the characteristic dilatation of the small blood vessels, whereas the neurasthenic felt the cold greatly and was often cold and clammy to the touch.

Again, the patient with Graves's disease was usually an optimist, while the neurasthenic was a pessimist. This was, as Plummer pointed out, easily illustrated when the thyreotoxic patient was asked to step up on to a chair. He tried confidently and failed because of his muscular weakness, whereas the neurasthenic, sure of failure, on being persuaded to try, stepped up quite easily. Realizing the general endocrine upset that was present in Graves's disease, it was easy to understand that cases were not infrequently labelled as diabetes because glycogen storage was upset and sugar was present in the urine, and a blood sugar curve showed a true Langerhans type of deficiency.

In doubtful cases Dr. Sewell had found the iodine test almost infallible, as the true thyreotoxic patient always got better rapidly on the administration of efficient doses of iodine, while the neurasthenic was unaffected. Single basal metabolic rate estimations were deprecated because of the possibility of error due to nervousness and bad breathing of the patients, but repeated tests were exceedingly valuable.

Further laboratory investigations had shown that in Graves's disease widespread metabolic disturbances were present. The high cholesterol and fat content of the blood showed a disturbance of fat metabolism, whereas calcium and phosphorus were excreted in greatly increased quantities and were lessened in the blood. In patients with high blood calcium and bony decalcification there was almost certainly an associated hyperparathyreoidism.

It was important to realize that these changes in metabolism were present, and to counteract them by treatment, and especially by increasing the caloric intake of the patient. Fortunately most of these associated disturbances of metabolism, whether secondary to the hyperthyreoidism or not, were largely influenced by the administration of iodine.

In these days of multiple laboratory tests it was increasingly important to balance their results by sound general clinical investigation and judgement. In Dr. Sewell's opinion many cases of toxic nodular goître were also overlooked, and this so easily occurred because the goître had usually been present for many years and the onset of toxic symptoms was often so very insidious. Such symptoms were often almost entirely cardio-vascular, and both the patient and his medical attendant failed to connect

the old-standing nodular goître with the cardiac disability. Accordingly, many cases went on to auricular fibrillation and some degree of congestive failure before the chief offender was blamed.

A careful examination of these patients, however, revealed many of the general symptoms of hyperthyreoidism already discussed, and an iodine test was usually conclusive.

In discussing ætiology, Dr. Sewell said that when Plummer published his work they had appeared to be much nearer to an understanding of the fundamental causes of hyperthyreoidism than they were today. As they were aware, Plummer differentiated Graves's disease definitely from toxic adenoma. He ascribed the former condition to a dysthyreoidism, the toxic factor being an excess of an incompletely iodized thyroxin, while the latter condition he held to be due to simple hyperthyreoidism due to the adenoma or adenomata becoming active producers of thyroxin.

Dr. Sewell thought that most people were now convinced that it was impossible to adhere to such an arbitrary distinction, because the one type merged insensibly into the other, and many cases of nodular goître became cases of Graves's disease, and as Dr. Wright Smith had just pointed out, it was quite impossible in most cases to differentiate microscopically the two types. It would be very nice and simple to accept the view that the full picture of Graves's disease was due simply to mass absorption from a fluid showing general hyperactivity, whereas the usually less severe symptoms of patients with adenomata were due to a much less massive absorption from only a patchily hyperactive thyreoid. Dr. Sewell found it difficult to accept such a view because he had seen quite a number of patients with severe nervous symptoms and exophthalmos with an insignificant goître and a comparatively slight rise in the basal metabolic rate. Such cases made it difficult to escape from the idea that a dysthyreoidism was present or that some other factor than the thyreoid condition was responsible for at least some of the symptoms. Aschoff held the view that some inherent condition of the nervous system was necessary before patients with hyperthyreoidism developed the full picture of Graves's disease. Such a view had much to recommend it. Oswald held the view that Graves's disease was due to an inherent thyreoid defect which led to its inability to take up and use iodine from the blood, although in the disease the blood iodine was actually raised, while the thyreoid iodine was greatly reduced. He would liken the function of the thyreoid in this connexion to the glycogenic function of the liver. Such a view was provocative of thought, but did not in any way explain the symptomatology of the disease. Scott Williamson's view was most attractive. He considered that the thyroxin was secreted by the thyreoid, passed by way of the sinusoid to the lymph vessels and was then carried to the thymus, where it was stored and possibly further elaborated. Scott Williamson held that in Graves's disease the cellular hypertrophy and hyperplasia partially blocked the sinusoid so that the immature thyroxin was directly absorbed by the blood vessels, as was the bile in a case of catarrhal jaundice. In this way he looked upon the symptom of Graves's disease as being really due to a dysthyreoidism. However, his work had not been yet confirmed by other observers. Marine took the view that the primary lesion in Graves's disease was due to deficient activity of the adrenal cortex which produced a secretion which had a controlling and steadying effect upon thyroxin production. He had much experimental evidence in favour of his view, and claimed to have successfully treated many cases of Graves's disease with a glycerol extract of adrenal cortex. Knowing as they did that there was a profound general endocrine and metabolic upset, Marine's work held much hope for the future management of this disease. However, in the present state of their knowledge of these diseases, Dr. Sewell felt that, as a physician, he was bound to advise all patients with severe symptoms to have surgical treatment because of the risk of permanent nervous and cardiovascular instability if the disease was allowed to persist for more than a limited time. Such cases could now be tackled surgically almost without risk if iodine were administered in efficient dosage for two or three weeks before operation was undertaken. Digitalis should also be intelligently given before operation to patients with any sign of congestive heart failure. This was a disease in which intelligent cooperation between physician and surgeon was essential.

Dr. Sewell had strong opinions about the advisability of removing septic foci, tonsils, teeth, *et cetera*, before operating on the thyreoid. These might have been of some causal importance, but once the thyreoid had become grossly hypertrophic and hyperplastic, it was the outstanding danger to the patient's future health and should be dealt with first, while septic foci could be dealt with later and at leisure. In early and mild cases the position was quite different and with the use of iodine, rest, and attention to septic foci, the thyreoid condition would often completely subside and the patient's health be restored to normal. Drug treatment as a part of the plan was often most helpful in this type of case, but Dr. Sewell felt it was too slow in its action to merit its use in severe cases in which time was such an important factor if irreparable damage was to be avoided.

In the management after operation Dr. Sewell wished to stress the importance of continuing a small dose of iodine daily for some months if the number of cases of recurrence of symptoms was to be reduced. He wished also to point out that many patients after subtotal thyreoidectomy presented temporarily some symptoms of myxœdema and it helped the thyreoid tissue to take up its work more quickly and adequately if small doses of thyreoid extract were given. This was usually necessary only for a short time, but its administration gave material comfort to the patient in that it quickly relieved the symptoms.

DR. SYDNEY PERN expressed interest in the views of the speakers, but found himself not in complete agreement with them. For satisfactory treatment of any disease, Dr. Pern considered the causation should be well known. Nothing had been heard of causation that evening, and, in his opinion, the causation of goître had nothing to do with lack of iodine. He had recently received a letter from Colonel McCarrison, who said, that he (the writer) was glad that Dr. Pern had opposed the iodine theory and had maintained the attitude that focal sepsis should be removed.

During a considerable experience in Gippsland twenty-five years previously, and later in the out-patient department of the Melbourne Hospital, he (Dr. Pern) had come to the conclusion that infection was the main causative factor in toxic goître. In his own practice he dealt with more patients suffering from goître than any other disease, and he had very few operations performed. By a process of culturing the tissue of goîtrous thyreoid glands organisms were obtained in every case. When removal of focal sepsis failed to relieve the goître, then he concluded that some other septic focus must remain undetected, and if such a focus were found but could not be removed, then an operation might be necessary. He had known patients whose thyreoid glands had been removed for the relief of exophthalmic goître without success, and later the exophthalmos had been cured by the removal of focal sepsis.

DR. K. D. FAIRLEY thanked the speakers for their interesting presentation of a fascinating subject. There was no doubt that a single so-called adenoma might be responsible for toxic symptoms.

The spontaneous disappearance of toxic manifestations, such as auricular fibrillation, after the enucleation of an adenoma, was frequently seen. If the nodule produced symptoms by leading to hyperplasia in the surrounding tissue, it must be assumed that satisfactory resolution occurred after its removal. It seemed probable that in some cases at least the nodule *per se* was responsible for the thyreotoxicosis. Plummer had found a striking difference in the tendenecy to recurrence in the two types of goître. Among more than seven thousand patients followed for some years after their original operation, further surgical operation had been required for recurrent symptoms fifty times more frequently in the exophthalmic type, the incidence of reoperation being 6·5% in the exophthalmic and 0·12% in the toxic adenomatous goîtres.

This was remarkable because in many instances only an enucleation of an adenoma had been done, and, in a large percentage, adenomatous tissue remained *in situ*.

Injury to the recurrent laryngeal nerves at operation was more frequent than was generally realized. With an abductor paralysis, the voice was unaltered, for the affected cord remained in the mid-line position. For this reason, unless the larynx were examined as a routine early in the post-operative period, the incidence of these lesions would be greatly under-estimated. Among 353 patients treated surgically for toxic goître at the Melbourne Hospital in the past five years, about 10% showed evidence of injury to these nerves, and in approximately half of them the paralysis of the vocal cord was permanent.

The end results, when auricular fibrillation was a complication of goître before operation, were far better in those in whom the normal rhythm was restored. Quinidine was valuable when this did not occur spontaneously within a fortnight of operation. There was seldom any indication for the use of this toxic drug before this period had elapsed, and in those with gross cardiac failure before operation it might be held in reserve for a longer time. If one course of quinidine failed, it should be repeated about three months later.

Glycosuria was not uncommon in thyreotoxic conditions. When a true diabetes was complicated by hyperthyreoidism, the relief of the latter by surgical operation was usually followed by an amelioration of the diabetes.

The diagnosis of thyreotoxicosis in diabetes might be difficult, as many symptoms were common to both. The therapeutic response to iodine was often helpful in diagnosis. In every case of diabetes, and especially in diabetic acidosis, the possibility of a complicating thyreotoxicosis should be considered and appropriate treatment instituted if there were any suggestion of its presence.

Focal sepsis should be eradicated in those under medical or X ray treatment, but in more severe cases thyreoidectomy should precede any major operation for the elimination of focal sepsis.

Unsatisfactory results were obtained in most cases of exophthalmic goître with any operation less extensive than a subtotal thyreoidectomy, which was also indicated in toxic adenomatous goître with multiple nodules. When the condition of the patient was unsatisfactory after half the gland had been removed, further interference should of course be postponed. To obtain the best results prolonged post-operative observation was essential.

Those who required treatment for hypothyreoidism after operation were all extremely grateful for the relief that surgical operation had afforded. Dr. Wright Smith had referred to one patient who developed a huge goître in the space of two months. The basal metabolic rate was +69%. On the assumption that this rapid enlargement was not likely to be purely hyperplastic, and in the hope of reducing the size of the gland for the surgeon, five milligrammes of thyroxin were injected intravenously without the slightest apparent change in the clinical findings in the next few weeks. Iodine was then given and after a subtotal thyreoidectomy, at which the left vocal cord was paralysed, the hyperplasia in the gland was found to be so intense that a diagnosis of carcinoma was seriously considered.

The most common difficulty in the diagnosis of toxic goître was presented by the patient with a simple goître, and a superadded cardio-neurosis or effort syndrome. The absence of a warm moist skin, failure to lose weight, and the disappearance of the tachycardia during sleep were clinical features against the diagnosis of thyreotoxicosis. A single estimation of the basal metabolic rate was often of little help. In the most difficult cases repeated metabolic estimations in conjunction with clinical observation over a definite period, and, finally, the therapeutic response to iodine might be necessary to establish a correct diagnosis. The history of one patient who was subjected to a subtotal thyreoidectomy because of a faulty diagnosis of exophthalmic goître was given to illustrate this diagnostic difficulty.

DR. W. OSTERMEYER pointed out that there had been insufficient stress laid on the value of long continued iodine administration. There was a suggestion that the cause of goître was not focal sepsis, but an endocrine upset. He had known a patient who had apparently been cured of the toxic symptoms of exophthalmic goître after long continued administration of thyreoid extract, though the exophthalmos had remained. The subject obviously called for further intensive study.

DR. KEITH HALLAM said that it was important to remember the usefulness of X ray irradiation in the treatment of toxic goître. It was useless in colloid goître and in toxic adenoma. It was most useful in the early stages of hyperthyreoidism, typically in the young woman with some enlargement of the thyreoid gland, who complained of being easily tired.

Small repeated doses of X rays were beneficial to many such patients, especially in England where it was less easy to arrange for operation. The results of this treatment showed between 50% and 55% of cures, 20% to 25% of patients whose conditions were ameliorated, and the rest failures. Careful control of dosage was required, otherwise superficial telangiectasis might result leading to disfigurement.

Mild irradiation of the patient's neck after thyreoidectomy would give great benefit in many cases. The type of large columnar cell in thyreotoxic goître was intermediate between the primitive or malignant cell and the normal cell. The occurrence of fibrosis due to irradiation hampering subsequent operation had been definitely disproved. Gain in weight was the chief obvious benefit in patients irradiated.

DR. W. KENT HUGHES said that in his opinion the removal of focal sepsis would improve the patient's condition, but in his forty years' experience he had never known it to cure exophthalmic goître.

DR. W. J. NEWING asked Dr. Sewell if he suggested operation in patients with no thyreoid enlargement but an increased metabolic rate. This rule had been followed in Plummer's clinic and the operation in a large number of cases had been followed by mild myxœdema.

DR. BALCOMBE QUICK expressed his thanks to the readers of the papers. He joined issue with Dr. Julian Smith in the latter's advocacy of the treatment of focal sepsis before operation on the goître, except in early and mild cases as indicated by Dr. Fairley. Otherwise he (Dr. Quick) preferred to deal with the thyreoid gland first, leaving the major or minor cause of focal sepsis for treatment later if practicable. He looked on Dunhill as a prophet rather than to Plummer, and he considered that the method of attempting to classify various types of goître into watertight compartments should be discarded. He considered that Dunhill's last paper was a masterly exposition of the subject.

Patients operated on for definite toxic adenoma could cause confusion by suffering an intense post-operative thyreotoxic crisis, and these patients should have a careful pre-operative iodine preparation. There was a dangerous significance in pre-operative vomiting, and such patients usually needed post-operative intravenous administration of glucose and continuous oxygen therapy which was of great value.

DR. VICTOR HURLEY expressed the thanks of the meeting to the speakers.

In reply to the discussion Dr. Sewell said that in diagnosis it was difficult to distinguish between the thyreotoxic patient and the neurasthenic patient with an effort syndrome. The latter had a cold dry skin and tended to be fat and flabby. The slight stare and the increased basal metabolic rate were of value in recognizing the case of early exophthalmic goître, and lastly, the effect of the illness on the nervous system should be considered. The neurasthenic was usually depressed and miserable, but the thyreotoxic patient was usually optimistic, irritable and restless.

Owing to the results of successful surgical operation the severe types of thyreotoxic goître were seldom seen now in Victoria, with auricular fibrillation, congestive heart failure, and lasting ill effects on the vegetative and central nervous systems, which never recovered completely even if a normal basal metabolic rate were restored. There was a stage in the evolution of thyreotoxic goître in which some patients might be cured by small doses of iodine, possibly aided by irradiation, but even in these cases operation should be advised if the condition were not much improved within two or three

months. It was hardly fair to say that Plummer operated purely on the basis of the basal metabolic rate as he also carefully considered the effects on the nervous system.

Dr. Sewell gave the details of a case of acute thyreotoxic goître in which the crisis was relieved by the intravenous injection of a solution of sodium iodide. Operation followed and the patient was restored to normal health and weight. Operation should be performed, probably within a month of the commencement of iodine therapy. In mild cases it was preferable to treat focal sepsis before the thyreoidectomy, in severe cases after the operation. Dr. Sewell expressed the belief that focal sepsis played a small part in the causation of thyreotoxic goître. Inability of the thyreoid gland cells to deal with iodine adequately was a more likely explanation.

Dr. Julian Smith, in reply, said that he was interested in Dr. Fairley's figures of the incidence of recurrent laryngeal nerve palsy. He felt that there were a larger number of injuries to these nerves than most people were willing to admit. Windy phonation was a common sequel of thyreoidectomy, and one singer he had operated on had suffered from a unilateral laryngeal paralysis, but recovered, and later sang well again. He agreed with Dr. Balcombe Quick as to the value of intravenously administered glucose, and he felt that their disagreement as to the time for dealing with focal sepsis was more apparent than real as each case had to be judged on its merits.

A MEETING OF THE SECTION OF NEUROLOGY AND PSYCHIATRY OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on April 21, 1932, DR. A. W. CAMPBELL in the chair.

General Paralysis of the Insane.

DR. G. P. U. PRIOR read a paper entitled: "Diathermy in the Treatment of General Paralysis of the Insane" (see page 882).

DR. CLIFFORD HENRY read a paper entitled: "The Routine Practice of Malaria Therapy" (see page 888).

PROFESSOR W. S. DAWSON mentioned the method of producing pyrexia by immersing the patient in a hot bath, as described by Gibson and Gordon in *The British Medical Journal*. He demonstrated graphs to show the much better results and smaller death rate after malaria therapy. There was an extraordinary variability in the number of hospital admissions for different periods, hence great difficulty in interpreting the results of therapy. The definitely reduced admission and death rate from 1922 onwards shown in English statistics was attributed by Professor Robertson to the introduction of "Salvarsan" ten years previously. He asked how far the blood changes noted by Dr. Prior might be due to concentration from sweating. He also wished to emphasize the great desirability of applying cardiac tonics such as digitalis and strychnine during the malarial treatment. Dr. Henry had given the impression that malaria treatment was unduly elaborate and needed many biological tests. But observation of the general condition was usually sufficient and it was wise to terminate the pyrexia if it was unsatisfactory.

DR. L. H. HUGHES said he was interested to hear Dr. Henry's suggestion as to what actually caused improvement in malaria therapy, namely stimulation of the reticuloendothelial system. It was fairly generally agreed that, while general nervous diseases were commonly found among tropical peoples, *tabes dorsalis* and general paralysis of the insane were very rare in the tropics. He also agreed with Dr. Henry as to the importance of testing the urine for urobilin. He was struck with the comparative ease with which these attacks were terminated, in contrast to his experience in ordinary malaria. "Plasmoquine" was a new preparation which was more effective if used with quinine.

DR. A. DAVIDSON said that the discussion was of particular interest, as general paralysis of the insane had been regarded as a hopeless disease. Previously it had been noted that a sudden disappearance of symptoms occurred after an attack of influenza. Discussion seemed to bear on the production of body temperature and this high temperature seemed to have the effect of killing infection. He felt that diathermy treatment was more practical from a general health point of view than malaria.

DR. O. LATHAM said that he wished to point out the difficulties facing an explorer in a new field like that of the treatment of general paralysis by diathermy. He thought that Dr. Prior and his staff were lucky as well as skilful, in that there were no serious results. Their courage and enterprise were worthy of appreciation. The reason why such small doses of quinine were effective in artificially produced malaria was, that the sexual gametes failed to appear after being through many hosts. Therefore very little precaution was necessary. He then emphasized the difficulty of obtaining a pure strain of malaria parasite, and of being sure that there was not a double infection. He wished to allude to a fatal case, in which the doctor had thought that the malaria had not taken, as there was no rise of temperature; but the patient had actually been so badly infected that the blood was full of pigment. He had to admire Dr. Henry's courage in infecting all sorts of patients, but he thought some discretion should be used in the choice of cases. He stated that there could be definite clinical improvement without any serological improvement or biochemical improvement. Intravenous medication was only necessary in cases of double infection. The malignant parasite seemed to lie quiescent in the host, but when given to a new patient, the malignant parasite appeared at once.

DR. J. A. L. WALLACE said he greatly admired Dr. Prior's enterprise in introducing this new form of treatment. In his opinion there was a natural reaction to malaria, which was better than the artificial reaction to diathermy; but if the same results could be obtained by means of diathermy, this was a much more desirable form of treatment. Malaria therapy had been commenced by Dr. Ewan and himself towards the end of 1926 and they had had a very anxious time on account of mixed infection.

DR. A. W. CAMPBELL said that both papers were of extreme importance and dealt with a subject of great importance. As Dr. Davidson had hinted, those who were old enough remembered earlier days when a diagnosis of general paralysis of the insane had been tantamount to signing the patient's death warrant in a relatively short period. The benefits gained by the introduction of these pyogenic methods constituted one of the greatest triumphs in the whole field of therapeutics. He would like to ask a question or two regarding prognosis. Most writers stated what seemed to be common sense, that patients with advanced disease and those with complications such as alcoholism and old age, were unfavourable from the therapeutic point of view. Dr. Henry had said that all benefited. As regards patients who, prior to treatment, had strong serological reactions, he asked whether they admitted of a more favourable prognosis than those whose serum reacted slightly. Others maintained that cases of the expansive type were more favourable. He would like to ask if there were any changes in the physical state, such as disappearance of the Argyll-Robertson phenomena. Finally, he would say that he greatly appreciated the thoroughness with which both these workers had performed their task, and even Dr. Prior's work, young as it was, would compare favourably with that performed in other parts of the world.

Dr. Prior in reply said that he could not account for the blood changes. American workers had observed an increase in the white blood cells, and he a decrease. He had not observed any changes in the reaction of the pupils nor in the reflexes. American observers remarked that the hot bath treatment was not a practical method, as the patient became exhausted in two hours.

Dr. Henry, in reply to Dr. Hughes's question as to how the infection was so easily cured, said that the difficulty was to keep the strain going, as it tended to die out in the absence of the sexual phase of the life cycle. He had not found any relationship to prognosis in the serological reactions or type of the disease. He had not observed any improvement in the Argyll-Robertson pupils, though it was stated in the literature that such improvement did take place as a result of malaria therapy.

Hospitals.

RADIUM AND X RAYS.

A COMMITTEE consisting of representatives of the metropolitan hospitals in Melbourne and the Department of Natural Philosophy of the University of Melbourne, has drawn up a list of recommendations dealing with means of protection against the harmful influences of X rays and radium, and the conservation of radium. These recommendations are being sent to hospitals throughout the State of Victoria. The committee has requested the publication of the recommendations in these pages.

NOTICE.[1]

Dangers of Radium and X Rays.

Radiation from radium or X rays acting upon the skin may set up local irritation, even malignancy. Acting generally, it may affect the blood or the reproductive system. Its action is cumulative.

X Ray Precautions.

Lead gloves and apron should be worn while screening. Minimum intensity, aperture and the time of exposure should be used. The risk of damage to the skin depends on these factors.

Lead and lead-glass screens are provided. Protect yourself. On no account should a patient be approached while deep therapy treatment is in progress.

Adequate ventilation of the X ray room must be maintained.

Radium Precautions.

When handling radium or radon, work quickly. The source of radiation should be kept as far as possible from the body and always screened by lead. On no account should radium or radon containers be handled directly with the fingers. Long-handled wooden forceps or rubber-covered metal forceps are essential. All manipulations with radium or radon should be performed behind lead screens. In threading radium needles, a suitable lead-protected clip should be used. Radium or radon in the theatre should be kept screened by lead. In transporting radium or radon, a lead box fitted with a long handle will keep radiation as far as possible from the hands. Nurses and attendants should not remain in proximity to patients undergoing radium treatment any longer than is necessary.

All unskilled work with radium and radon should be carried out as far as possible by temporary workers. Permanent sisters must not handle radium apparatus.

General Precautions.

All persons engaged in X ray or radium work should submit themselves to blood examinations or other tests at periods not greater than three months.

Observe the rules of general health. When off duty spend as much time as possible in the open air.

Do not let familiarity make you careless.

Electrical Shock.—Contact with high tension leads will cause death. On no account should the X ray equipment be approached during operation. With a valve operated plant, the condensers should be discharged before the installation is touched. In the event of electrical shock, care should be taken in removing the victim from wires, and treatment, artificial respiration *et cetera*, should be commenced immediately. Rubber gloves or macintosh should be used to pull the patient away from the source of current or, failing this, a loop of dry rope or bent stick.

Artificial Respiration.

Artificial respiration in the treatment of a person who has suffered an electrical shock should be commenced immediately. It should not be assumed that death is present because signs of life are absent. Make certain that the air passages are not obstructed and clothing *et cetera* does not prevent the necessary expansion of the chest.

Adjust the Patient's Position.

At once lay the patient in a prone position (that is, back upwards), with arms extended above the head, and his head turned to one side, so as to keep his nose and mouth away from the ground. Do not waste time in loosening clothing. No pad is to be placed under the patient, nor need the tongue be drawn out, as it will fall naturally towards the lips.

To turn the patient to the prone position, stand at his side, grasp the clothing at the hip on the opposite side of the body and pull smartly over.

Imitate the Movements of Breathing.

(*a*) *Induce Expiration.*—Kneel at one side of or across the patient, facing his head, and place your hands over the lower ribs, the thumbs nearly parallel and close to the spine, the fingers slightly open and pointing towards the shoulders. Keeping your arms quite straight and rigid and leaning your body forward, slowly apply firm but not violent pressure downwards upon the lower part of the chest, thus driving air out and producing expiration.

(*b*) *Induce Inspiration.*—Draw back your body somewhat more rapidly and relax the pressure, but do not remove your hands; this produces inspiration.

Alternate these movements by a rhythmical swaying forwards and backwards of your body from the knee joints, twelve to fifteen times a minute.

General.—The above is a modification of Schaefer's method of artificial respiration.

While artificial respiration is being done, other useful steps may be employed to excite respiration, such as applying smelling salts to the nostrils, or flicking the patient with a wet towel. When natural breathing begins, regulate the artificial respiration to correspond with it, and promote circulation by rubbing the limbs energetically towards the heart.

Artificial respiration must be continued until respiration is restored or a doctor pronounces life to be extinct.

INSTRUCTIONS TO BE SENT TO HOSPITALS USING RADIUM AND X RAYS.

1. The enclosed notes showing the dangers of radium and X rays should be posted prominently throughout the departments of the hospital to which they apply. For example, they should appear in the out-patient department where radium is being used, in the wards in which radium patients are treated, in each diagnostic X ray room and in the deep therapy department.

2. The technicians working in the radium and X ray departments should be instructed verbally in the precautions to be taken and trained in the resuscitation of the electrically shocked.

3. Precautions to be taken in electrical equipment should include inspection of the insulation and care that insulators are kept dry and free from dust. All metal portions of the equipment with which the operator may come in contact, should be sufficiently earthed. Instructions should be given that, in the use of valve operated equipment, condensers should be discharged before equipment is approached, either after completion of a treatment or adjustments to plant.

4. Warning should be given as to the danger of fire in the use of ether or ethylene anæsthetics in relation to X ray plant.

5. Precautions of fireproof storage for X ray films with adequate ventilation and alarms should be established, the stock of unused X ray films being kept at the minimum compatible with efficiency.

6. All rooms containing X ray apparatus, particularly deep therapy plant, should be adequately ventilated. If natural ventilation is insufficient, artificial ventilation by electrical fans *et cetera*, should be installed.

7. All diagnostic X ray and deep therapy apparatus should be adequately protected by lead screens and

[1] Notice to be printed in bold type and placed in every room in which X ray or radium is in use.

observed only through lead-glass windows or by mirrors. The equipment of the department should be so arranged that the maximum protection should be given to technicians during the operation of the plant. Lead-glass windows should be provided for observation of deep therapy cases or, alternatively, a system of mirrors arranged so that patients may be observed without the technician receiving any radiation during the operation of the plant. The protection of diagnostic screening units should be approved by the committee set up by the conference, and should be tested at intervals.

9. The permanent sister or technician in radium work should handle radium as little as possible, all work being done by temporary workers, such as nurses, who should not work in the department for longer periods than three to six months. The permanent sister in charge should be regarded as an instructor and an administrative officer rather than part of the working staff of the hospital. All persons working with radium or X rays should have at least one month's holiday *per annum*, best divided into two periods of fourteen days each. The working hours of technicians and sisters should not exceed thirty-seven per week, arrangements being made to give half a day off per week in the middle of the week.

All workers in the diagnostic and therapeutic X ray and radium departments should have a blood examination on first commencing work in the department, and thereafter at three-monthly intervals. These reports should be kept by the officer in charge of the department, graphed, and reported to the medical superintendent of the hospital if any changes occur.

Damaged radium containers should be placed in a glass tube and hermetically sealed, and as soon as possible sent to the University for examination and advice.

Lead boxes are provided for carrying radium about the hospital with at least one centimetre of lead protection and with a leather strap attached, at least 45 centimetres (eighteen inches) in length to keep radium as far as possible away from the hands.

During sterilization of radium or radon, either the needle should be kept in a lead box or the sterilizer should be screened from the theatre by a lead sheet at least one centimetre in thickness.

The safes holding radium should be placed outside the ward or portion of the hospital in which technicians, nurses, *et cetera*, are working, and should be adequately protected by lead.

Suggested System of Radium Control for Hospitals.

At the present time, owing to exchange *et cetera*, each milligramme of radium is worth £20 in its container, and therefore detailed safeguards have been suggested.

The main principles for protection against loss of radium can be divided into: (1) storage of radium when out of use, (2) precautions to safeguard loss of radium on issue from the safe. This last may be divided into: (i) care of radium after issue from the safe, but not in actual use upon a patient; (ii) out-patient control of radium; (iii) ward control of radium; (iv) systems of control of whole of radium; (v) insurance of radium; (vi) practical points in the technique of use of radium.

Storage of Radium.

The essentials of an efficient system of storage are as follows:

(*a*) A safe for the bulk storage of hospital radium. This should be fireproof.

(*b*) Sufficient protection by lead, according to the requirements of the international regulations, depending upon the amount of radium stored.

(*c*) Within this safe should be arranged individual containers for each item of radium apparatus. This means that it should be unnecessary to remove a large number of needles from the safe to obtain the individual needle required. These containers are best arranged in the form of brass rods, having a hollow at one end to contain the needle, the rods fitting into a block of solid lead.

(*d*) The safe storing radium not in use should be well away from the wards or other rooms where radium workers are likely to be situated.

(*e*) Responsibility for radium in the bulk store should be confined to one officer, who is nominally responsible for the whole radium supply of the hospital irrespective of its situation, but who is personally responsible for the radium stored in the bulk safe.

Precautions to Safeguard Radium on Issue.

Care of Radium After Issue from the Safe, but not in Actual Use upon a Patient.

(*a*) Radium should not be out of bulk store for more than twenty-four hours unless it is actually in use for a patient. That is, radium required for the day should be issued only as necessary for that day's cases.

(*b*) After removal from the patient, radium should be returned to the bulk store as soon as possible, at least within twenty-four hours.

(*c*) When not in use and not in store, radium should be kept in a separate safe in the department to which it is on issue. This necessitates the provision of a safe in the ward, the out-patient department and the operating theatre.

The Responsibility for Radium on Issue.

1. The chief responsibility for all radium at the hospital rests with the medical officer in charge of the radium or other officer appointed by the committee.

2. This responsibility may be delegated to other officers according to the situation of the radium.

3. Radium issued by the medical officer from the bulk store becomes immediately the responsibility of the sister or other person accepting the radium. This person should check the needles received before leaving the safe.

4. Radium issued for out-patient use becomes the responsibility of the sister in charge of radium out-patients, who should store radium not actually in use in a safe in the out-patient department.

5. Radium issued to the theatre sister for use by surgeons in the theatre becomes the responsibility of the theatre sister until the patient returns to the ward with the radium inserted or, alternatively, until unused radium is returned to the bulk store.

6. The surgeon inserting radium needles is responsible for counting the needles inserted, and further should draw, or cause to have drawn by the assistant, at the time of insertion, a diagram showing the number and orientation of the needles inserted.

7. The theatre sister and the ward sister must check the number of needles during or after insertion into the patient.

8. The responsibility for the radium actually in a patient is delegated to the ward sister immediately the patient leaves the theatre, and remains her responsibility until the radium is removed and returned to the bulk store.

9. Radium removed in the ward should immediately be checked, cleaned and placed in the ward safe pending its return to the bulk store.

10. When the ward sister is off duty, the radium in patients in the ward and in the ward safe becomes the responsibility of the senior nurse on duty in the ward. This includes both day nurses and night nurses.

11. The nurse cleaning radium under the supervision of the sister in charge of the ward, is temporarily responsible for that radium until returned to and checked into the ward safe by the ward sister.

Out-patient Control of Radium.

Out-patients under treatment with radium should be under continuous indirect observation, or, alternatively, adequate steps should be taken to prevent the patient leaving the building unattended by a member of the radium staff.

Ward Control of Radium.

1. Where possible, all patients who have radium applied should be grouped in a special radium ward.

2. The staff of the radium ward should be instructed in the dangers and responsibilities of radium work.

3. Adequate precautions should be taken to prevent patients leaving this ward, and it is advisable that a patient

under radium treatment should be confined to bed and not permitted to go to bathrooms, lavatories or other rooms accessory to the ward.

4. Where feasible, radium wounds should be kept covered to prevent the patients interfering with the needles and threads.

5. Special bins should be provided in the wards to receive dressings from radium patients. A sufficient number of these of different colours should be available corresponding with the number of operating days per week. For example, dressings from a patient operated upon on Monday should only go into, say, a blue bin, which will not be emptied until all radium inserted on that day has been removed and checked. Disks of colour corresponding to the bins should be placed over each patient's bed, dressings from such patients only going to the corresponding coloured bin. These disks should bear a notice stating that radium is in use in this patient.

6. Dressings should be done only by senior nurses who have been warned of the dangers of loss of radium.

7. Bed linen *et cetera*, from radium patients should be examined before forwarding to the laundry, being shaken on to the floor to insure no needles are included.

8. Floor sweepings should be retained for at least one week in a two-bin system, one bin being in use for a week. At the end of this week the first bin is locked and the other placed in commission. At the end of the second week, if no radium has been lost, the first bin is emptied and the second locked, the first going into commission again.

9. Bed-pans, urinals *et cetera*, used by patients with radium in the pelvic regions should be carefully searched before being emptied into the sewerage system. A sedimentation tank is desirable for the radium ward.

Systems of Control of Whole of Radium.

The principle of the control system for all radium in the hospital should be as simple as is possible with efficiency. As little as possible actual writing should be necessary, and the system should also, as far as possible, be free from risk of error due to personal factors. Any stock of radium in a hospital should be most easily controlled by the token system detailed below.

The principle of the token system is that each item of radium is represented by a suitable token, and the movements of radium within the hospital are shown by the movements of tokens on various token boards.

1. *Control of Whole Radium Supply.*—A master token board indicates the movements of all radium in the hospital.

2. *Ward.*—If all radium cases are in one ward, one token board should be situated in proximity to the ward and under the charge of the ward sister or her deputy.

3. *Theatre.*—A theatre token board should be under the control of the theatre sister responsible for radium in the theatres.

4. *Out-patients.*—An out-patient token board should be situated in the out-patient department in charge of the out-patient sister.

If radium is in use in several wards, boards should be provided in each ward, the tokens being stamped with the ward number only, so that a token hanging on the master board in its correct place will show that the particular piece of radium in question is in the ward indicated by the token. In the ward there will be spaces on the token board corresponding to the number of pieces of radium in the wards. In each ward an adequate number of tokens for ordinary working purposes is required.

The tokens in each situation, that is master, theatre, ward or out-patient department, should be of different shapes. Each piece of radium apparatus should be represented on every board in the hospital by tokens of different shape.

Token boards should be situated behind locked glass doors and the token board key held only by the person responsible for the radium in each department.

At the safe, a blank board is hung, on which are placed master board tokens corresponding to all the radium on issue.

Radium is issued from the main safe only on presentation of master board tokens corresponding to the required radium.

Master board tokens are issued to the sister requiring radium in exchange for her own tokens corresponding to each piece of radium, which then are hung on the master board in place of the master board tokens. The master board thus indicates not only where the radium is situated, but also which sister is responsible for the radium. The master board, therefore, indicates: (i) the remaining radium available in the safe for use, shown by master board tokens on the master board; (ii) the radium on issue to the theatre, shown by theatre tokens on the master board; (iii) the radium in patients in the ward, shown by ward tokens on the master board; (iv) the radium in use in the out-patient department, shown by out-patient tokens on the board. This master board must always be complete. Immediately a token is removed from the master board, one corresponding token must be replaced indicating the situation of and the responsibility for the radium.

When a patient with radium is taken from the theatre to the ward, the ward sister automatically assumes responsibility and gives to the theatre sister tokens corresponding to the radium in use. These are exchanged at the master board by the theatre sister, who gets her own tokens back. The ward tokens are then hung on the master board, indicating the changed situation of the radium.

Radium after removal from the patient in the ward is returned to the main safe by the ward sister, who receives in exchange corresponding master board tokens. These are exchanged for her own tokens on presentation at the master board and blanks on her own board are obliterated.

Radium in the safe can be checked against the safe board and the master board by examining tokens and radium. Radium in the ward can be checked by examining the ward radium book and counting the spaces on the ward token board.

Record books should be kept in ward, out-patient department and theatre, recording names *et cetera*, and details of radium used on each patient. No record book is necessary at the main safe.

No signing is necessary for any radium. Radium is issued and exchanged only against the appropriate tokens, and full responsibility must be accepted by all concerned for accurate working of the system. Each responsible person is directly liable either for the piece of radium apparatus or the possession of a token corresponding to it.

The respective sister or her deputy is responsible for all radium under her care as shown by her own board.

The medical officer in charge is responsible for the radium in the main safe. The master board should be under the control of some other responsible person, so that radium is issued only after the transaction has involved three people.

Insurance of Radium.

It is advisable that all radium and radium containers should be insured against loss or destruction. Alternatively, a sinking fund should be established sufficient to cover any losses and replacements. The decision as to which of the above practices should be followed should be at the discretion of the hospital.

Practical Points in the Use of Radium.

1. Where it is considered that there is a possibility of loss of radium, radon should be used in place of radium needles.

2. Needles should be threaded with linen thread (Pagenstecher).

5. If there is a risk of displacement of a needle, it shown be sewn *in situ* by some suitable method.

4. If needles are being sewn in, the threads should be tied together by a reef knot at the eye of the needle, and, after suturing in, the final knot should be tied as near as possible to the eye of the needle.

5. On external lesions the threads of needles placed in opposite directions should be tied together to prevent displacement.

6. As many needles as possible should be tied together in groups, the strings from each group being strapped to the skin some distance from the wound.
7. In vaginal and uterine cases, the vagina should be packed with iodoform gauze and a silkworm gut suture placed across the vulva to prevent loss of packing. All threads from radium needles should be tied to this suture and the threads in turn strapped to the groin.
8. Non-residual diet is advised in all external genital and rectal cases.
9. In using an introducer for radium needles, care should be taken not to injure the thread of the needle by partially cutting it with the introducer.

Correspondence.

OBSTETRICAL RADIOGRAPHY.

SIR: I am sure Dr. Lethbridge has quite misunderstood my previous letter, as it was never suggested that X ray investigation should supplant any of the ordinary and very necessary clinical examinations, but has been advocated only as an additional means of diagnosis when other methods are insufficient to provide all the information desired. No attempt has been made to discourage other methods of examination, but their limitations should be appreciated.

There is not the slightest doubt about the "bogy", and as this aspect has been dealt with very fully in my article, already quoted, it is superfluous to repeat it here.

My reasons for calling Dr. Davidson's criticisms unfair are:

(*a*) "If it is known that large doses result in microcephaly *et cetera*, it is reasonable to fear that small doses may result in less damage." If it is known that a jump from a height of twenty or thirty feet may cause serious injury, it is just as reasonable to fear that a jump of a couple of inches may result in less gross damage!

(*b*) "Murphy goes so far as to say that 'if a growing embryo is unwittingly exposed to irradiation, and its existence discovered later on, the pregnancy should be terminated'." There is not the slightest doubt that Murphy meant exposure to therapeutic doses and not to the minute fraction of this required for diagnosis.

(*c*) "There are cases in which an apparently normal child physically is found before the age of five years to be imbecile or mentally defective to a mild degree because of birth trauma to the cranium undiagnosed at the time of birth" *et cetera*. My article referred to was written six years ago and obstetrical radiography was performed for several years before that, yet there is no evidence whatever anywhere, despite the lapse of many years, of injury to the fœtus from diagnostic procedures, despite the critical observations of many competent observers.

(*d*) "The difficulty of rightly interpreting such a tiny feature as the ossification centre makes this method of diagnosis academic rather than practical." The epiphyses for the distal ends of the femora and proximal ends of the tibiæ are not so tiny as to prevent recognition in all good Röntgenographs. Admittedly poor films, taken by inadequate equipment and poor technique, are useless; but the epiphyses of the femora and tibiæ have been repeatedly identified during the later months of pregnancy. These are the first epiphyses in the body to appear.

(*e*) Regarding the remarks concerning the overlapping fœtal bones, which have been so frequently demonstrated as indicating the death of the fœtus, has Dr. Davidson (or anybody else) ever met this sign in the presence of a living fœtus?

(*f*) "One patient was diagnosed by a very capable radiographer from very clear films as having above the average diameters." Was not the radiologist correct? Surely Dr. Davidson did not expect him to diagnose abnormal resistance of the soft parts! This is surely not a reason against the employment of radiological pelvimetry and in any case clinical pelvimetry had never been discouraged.

(*g*) Even in quoting my own remarks, in his letter of May 28, Dr. Davidson has not appreciated the fact that the apparent absence of physical and mental abnormality in children has followed some intrauterine therapeutic doses. If, then, some fœtuses are (apparently at least) able to stand a therapeutic dose without injury, what possibility is there of any injury whatever following exposure to the infinitely smaller diagnostic dose?

Yours, etc.,

H. FLECKER.

Cairns.
June 3, 1932.

"AVERTIN."

SIR: It is evident from Dr. Balaam's letter, and articles recently published in the lay Press, that the leader in your issue of May 21 last, headed "Avertin", has been accepted by some readers as expressing an unfavourable review of the drug. A more careful analysis of the statements contained therein, however, shows that this was certainly not the intention of the writer, but was merely meant as a plea for the continuance of basal or slightly supra-basal doses as a maximum, and an invitation to practitioners and hospitals to publish full details of any fatalities which may occur, especially of those, if any, in which the medical and surgical risks have been small. Both these laudable objects will be heartily endorsed. "Avertin" has everywhere met with an unusually critical reception, but so much investigation of the pharmacology and risks intrinsic to the drug itself has now been done, however, that Australian practitioners may continue its use as a basal narcotic in perfect confidence, undismayed by any local opinion or accident. The drug, like the barbiturates, has won for itself a permanent place in the equipment of the modern anæsthetist and will continue to confer its boon increasingly until the humane concept of basal narcosis becomes universally accepted.

Yours, etc.,

KEMPSON MADDOX.

Sydney,
June 7, 1932.

MENTAL DISORDER.

SIR: Would it be possible, through the columns of your paper, to draw the attention of general practitioners and those responsible for the training of medical students to the report of the Council of the British Medical Association appearing as a supplement to *The British Medical Journal*, April 30, 1932? Otherwise it may not have the attention it deserves.

In this report there is a very valuable statement regarding the relationship of the private practitioner to mental disorder. It embodies the views and recommendations of most of those interested in filling the gaps which exist in the treatment of mental disorders, and in the training of the medical student and post-graduate.

I should like to quote from this report at length to show the enlightened attitude of those responsible, but will content myself with the following paragraph:

"It has recently been recognized that many of the emotional disturbances, changes or abnormalities in character or conduct, as well as certain physical symptoms, are due to mental disorders, and that these disorders evince themselves in a considerable proportion of the patients daily seen by the private practitioner."

Trusting that attention given this report will help to remove the discredit at present attaching to the medical profession in this respect.

Yours, etc.,

JOHN F. WILLIAMS,
Psychiatrist to the Children's and Saint Vincent's Hospitals, Melbourne.

June 9, 1932.

Post-Graduate Work.

POST-GRADUATE WORK IN PERTH.

THE Western Australian Branch of the British Medical Association announces that the annual post-graduate course will be held in Perth from July 11 to July 16, 1932. The course commences at 9.30 a.m. on July 11 and terminates on the morning of July 16.

Dr. Victor Hurley and Dr. L. E. Hurley, of Melbourne, have kindly accepted the Post-Graduate Committee's invitation to be their guests and will be the principal lecturers.

The subscription to the post-graduate course is £2 2s., and application for all information should be addressed to Dr. J. G. Hislop, Honorary Secretary for the Post-Graduate Committee, 260, St. George's Terrace, Perth.

The annual dinner of the Western Australian Branch will be held on the evening of July 16, 1932.

Books Received.

SPIRITUALISM FOR THE ENQUIRER, by H. E. Hunt; 1931. London: Rider and Company; Melbourne: Robertson and Mullens. Foolscap 8vo., pp. 158. Price: 4s. 3d. net.

A THOUSAND MARRIAGES: A MEDICAL STUDY OF SEX ADJUSTMENT, by R. L. Dickinson and L. Beam, with a foreword by Havelock Ellis; 1932. London: Williams and Norgate; Baillière, Tindall and Cox. Demy 8vo., pp. 507. Price: 21s. net.

THE PRACTICAL MEDICINE SERIES: NEUROLOGY AND PSYCHIATRY; Series 1931. Chicago: The Year Book Publishers. Crown 8vo., pp. 471. Price: $2.25 net.

THE PRACTICAL MEDICINE SERIES: DERMATOLOGY AND SYPHILIS; UROLOGY; Series 1931. Chicago: The Year Book Publishers. Crown 8vo., pp. 472. Price: $2.25 net.

TEXTBOOK OF MEDICINE BY VARIOUS AUTHORS. Edited by J. J. Conybeare, M.C., M.D., F.R.C.P.; Second Edition; 1932. Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 1023, with illustrations. Price: 21s. net.

THE PRACTICE OF CONTRACEPTION: AN INTERNATIONAL SYMPOSIUM AND SURVEY, Edited by M. Sanger and H. M. Stone, M.D., with foreword by R. L. Dickinson, M.D.; 1931. London: Baillière, Tindall and Cox. Royal 8vo., pp. 334, with 22 illustrations. Price: 21s. net.

THE CRITICAL AGE OF WOMAN, by W. M. Gallichan; 1932. London: Noel Douglas. Crown 8vo., pp. 160. Price: 4s. 6d. net.

A NEW THEORY OF CANCER AND ITS TREATMENT, by C. F. Marshall, M.Sc., M.D., F.R.C.S.; 1932. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 53. Price: 3s. 6d. net.

Diary for the Month.

JUNE 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 30.—South Australian Branch, B.M.A.: Branch.
JUNE 30.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Resident Medical Officer (male).

LEWISHAM HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Physician.

MATER MISERICORDIÆ PUBLIC HOSPITAL, QUEENSLAND: Resident Medical Officer (male).

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

SAINT VINCENT'S HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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